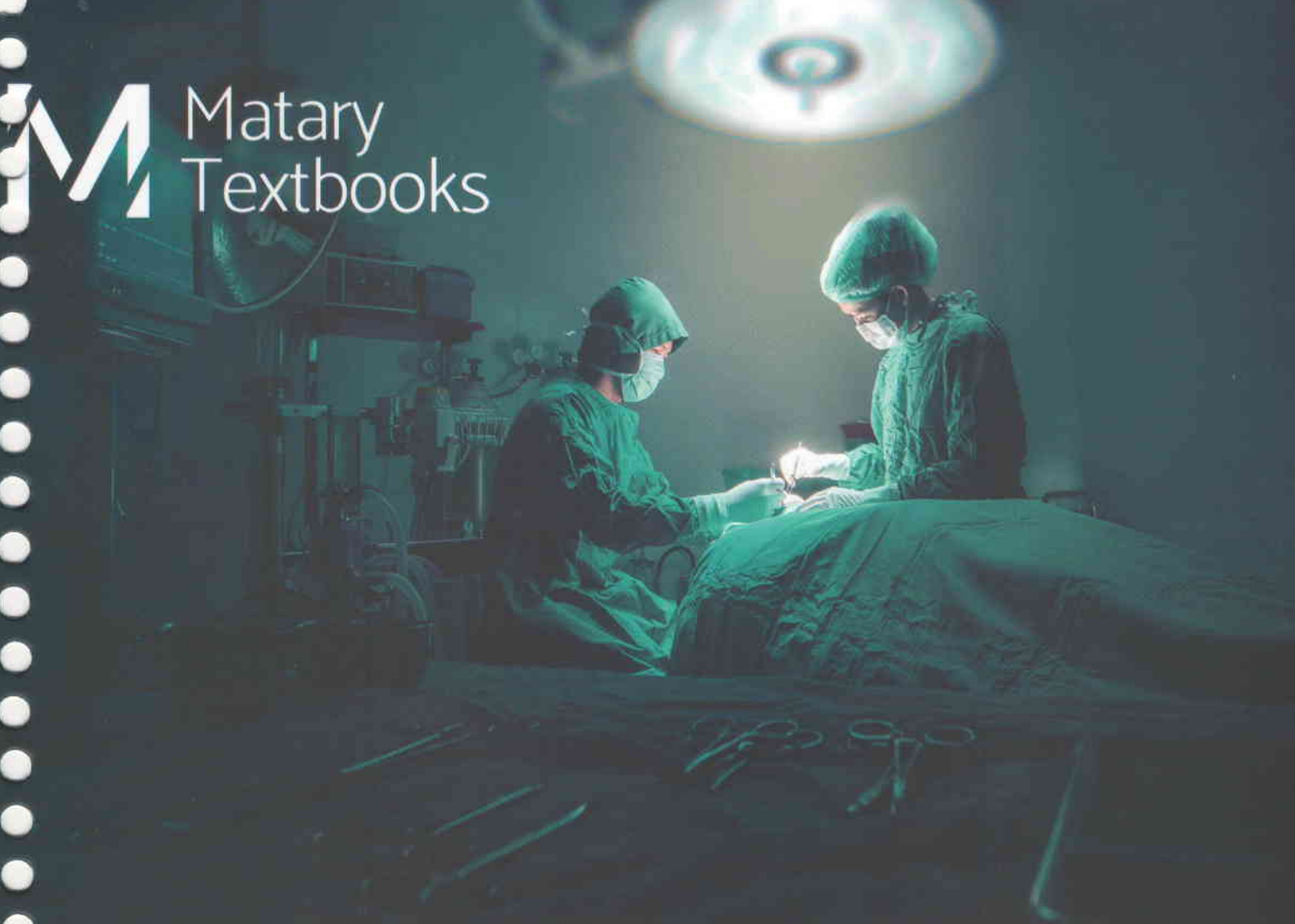




GENERAL SURGERY

VISIT...

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Caliente.COM

Acknowledgement

I'd like to thank my team who have worked to let this version of the book come to light..

Edited by

Ahmed Ibrahim Salim

M.B.,Ch.B

Cairo University

Designed by

Khaled Daif Allah

M.B.,Ch.B

Cairo University

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PREFACE

This book provides an update for medical students who need to keep abreast of recent developments. I hope also will be useful for those preparing for postgraduate examination.

This book is designed to provide a concise summary of General surgery, which medical students and others can use as study guide by itself or with readings in current textbooks, monographs, and reviews.

The author is extremely grateful to all the contributors for the high standard of the new chapters, and hopes that you the reader, will enjoy going through these pages as much as he had.

M. El-Matary

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Wounds



- Wound is forcible loss of continuity of soft tissues by mechanical trauma.
- Types Of wounds are **Open** or **Closed**.

Lacerated wounds



- Caused by severe violence with blunt heavy objects e.g RTA.
- They are irregular & the tissues are severely traumatized & devascularized.
- Inflammatory edema develops after a few hours → ↑ tension inside the wound → 2nd ischemia.
- They are usually contaminated → ↑ risk of infection.
- They are commonly accompanied by a degloving injury (skin & SC fat are degloved).

Abrasions



- Scraping away of skin superficial layers due to friction with rough surfaces.
- The wound is very painful due to exposure of the sensitive nerve endings.
- They require cleaning with a bland antiseptic & a non adherent dressing.

Incised wounds



- Due to sharp objects as e.g razor or knife
- The wound is longer than deep, its edges are clean cut & there is no much tissue damage.
- There is usually extensive hge. Tendons & nerves are liable to be cut.

Missile wounds



- They are very serious as the bullet transmits its high kinetic energy to the tissues.
- The kinetic energy is calculated by the formula (kinetic energy= $MV^2/2g$).
- So the high velocity missiles are much more serious than low velocity ones (pistols)
- The damage is due to the following factors:
 - a. Direct damage by missile track.
 - b. Shock waves preceding missile injury to the body.
 - c. Temporary cavitational effect.
 - d. If bone affection; bone fragments act as 2nd missiles → more damage.

Hematoma



- It occurs if the amount of bleeding is excessive.
- At 1st, it's cystic → clot within hours → liquefy later.
- If it's small → aspirate by wide-bore cannula.
- If it's large → surgical evacuation under aseptic condition.

Penetrating wounds



- Caused by pointed objects (nails & daggers).
- The wound is more deep than long → ↑ risk of injury to deep important structures.
- The external opening is small → ↓ drainage → ↑ risk of infection.
- They are deceiving so the surgeon may suture the wound & miss an injured viscus inside.

Contusion (Ecchymosis)



- Blunt trauma → extravasation of bl. from injured capillaries without swelling formation.
- The injured area becomes painful & swollen.
- The color is at 1st bluish (the extravasated bl.) the turns into brownish or green.
- If elevation occurs, minimize edema & apply local anti-inflammatory ointment.

Bites



- Either human or animal bites. They are lacerated & contaminated with high risk of infection.

Management of open wounds

1. Inquire about the time & cause of injury.
2. Prophylaxis against tetanus.
3. Prophylactic antibiotics. In deep or lacerated wound → add antibiotics against anaerobic infections.
4. Control bleeding by direct local compression by sterile dressing & tight bandage.
5. Don't apply tourniquet except as temporary measure before taking the pt to the theatre.
6. If fracture is suspected → arrange for x-ray & splint the fracture.
7. If pulses are not palpable after ttt of shock → duplex is mandatory.

Management in the theatre:

1. Clean the wound by saline irrigation, remove the FBs & sterilize it by antiseptics e.g bovidone iodine (betadine).
2. Repair any vascular injury acc. to the usual principles.
3. Repair any tendon or nerve injury in the clean incised wounds. In contaminated wounds, it's better to delay the repair.
4. Repair any ms injury by mattress sutures.
5. Leave the fascia opened in contaminated wounds or if there is extensive tissue destruction.
6. Skin:
 - Clean & early (<6 hrs) → 1^{ry} closure.
 - Contaminated or late → delayed 1^{ry} closure.
 - Lacerated → debridement to leave healthy vascularized tissues.
 - Degloved skin injury (skin viability is doubtful) →
 - a. Delay the decision till the 2nd look after 3-4 days.
 - b. The deep fascia & skin should be left open.
 - c. If there is a skin defect, pt will be reassessed when the tissues are healthy then skin graft or flap may be performed.
 - Associated fracture with possible infection → external fixation.
 - Missile injuries → exploration.



In contaminated wounds

- Don't do nerve or tendon repair.
- Don't close the deep fascia or skin.

1. Age: Wound healing is slow in elderly persons due to a reduced rate of protein turnover.

Components of wound healing

1. **Wound contraction**
(Starts immediately after wounding till 2-3 weeks due to special myofibroblasts containing actin): helps to diminish the size of wound.
2. **Granulation tissue formation** which is replaced later by fibrous tissue.
3. **Epithelization.**

Clean sutured wounds epithelialize in about 2 days. Contraction & granulation tissue/fibrosis occur to a little extent & later they have a larger contribution with gaped wound edges to fill the gap then epithelialization follows.



N.B:
Cells: platelets, PMNLs, macrophages, lymphocytes, fibroblasts, endothelial cells & epithelial cells.
Growth factors & cytokines: PDGF, TNF- α , IL-1 & FGF.

Stages of wound healing: «Overlapping»

1. Hemostasis & Inflammation phase:

- Injury of blood vessels → aggregation of platelets & activation of coagulation.
- This is followed by chemotaxis of leucocytes, macrophages & lymphocytes due to cytokine & GFs release by the platelets.
- Macrophages play an important role in phagocytosis & wound debridement through GFs release → Fibroblast & Endothelial cell recruitment & activation.
- This phase lasts for about 5 days, but it may be prolonged if there is wound infection.

2. Proliferation phase :

- This phase is characterized by the proliferation of fibroblasts from the surrounding tissues to secrete collagen fibers.
- Endothelial cells proliferate from intact venules to form new capillary buds with fibroblasts form the granulation tissue.
- Epithelial cells proliferation from wound edges. Young epithelium is thinner & possesses less sweat & sebaceous glands & hair follicles.

3. Maturation & remodeling phase:

- Deposition of the collagen fibers (III then I) then become thicker & arranged along the lines of stress → ↑↑ the tensile strength of the wound.
- The process of remodeling continues for about one year.
- A wound never attains its original tensile strength.

In addition to the above mechanisms, wound contraction helps to diminish the size of wound.

Types of wound healing

- 1st intention** in clean wounds when they are immediately closed by sutures /clips. It occurs with minimal scarring → nice neat scar.
- 2nd intention** when the wound edges aren't approximated or when gaping occurs as a result of a hematoma or wound infection. It occurs by infilling with granulation tissue and so there is more fibrous tissue → ugly scar.
- 3rd intention** contaminated wounds are left open for about 5 days. If there are no signs of infection; delayed 1st sutures can be performed.

2. Nutritional state:

- Protein deficiency → ↓↓ collagen synthesis.
- Vitamin C deficiency is responsible for lack of maturation of procollagen.
- Vitamin A deficiency → deficient epithelialization.
- Ca, zinc, Cu & Mg also affect wound healing.

3. Debilitating diseases: Uremia, jaundice, cirrhosis, DM & malignancy delay wound healing.

4. Drugs: Steroids and cancer chemotherapy & immunosuppressive drugs impair wound healing.

II- Local factors:

1. Vascularity: A good blood supply (e.g. in the face & scalp) → nice healing while a poor blood supply (wounds below the knee) → delayed healing.

2. Immobilization: movement damages the bl. supply → delay healing.

3. Irradiation → arteritis obliterans → Ischemia → ↓ wound contraction & granulation tissue format.

4. Tension: ↑↑ tension e.g. sutures under tension, hematoma, infection → ischemia & impaired healing

5. Infection: Fibroblasts compete with bacteria for oxygen & nutrition. Moreover, bacteria secrete collagenolytic enzymes, which destroy collagen fibers.

6. FBs & necrotic tissue: impair wound healing.

7. Adhesion of wound to a bony surface: Prevents wound contraction e.g. chronic venous ulcers.

8. Impaired venous drainage: as in postphlebotic limbs impairs wound healing.

Complications of wound healing

1. Wound failure (wound dehiscence): General or local factors which affect wound healing → wound failure. Failure of an abdominal wound is called burst abdomen.

2. Stretching of the scar.

3. Hypertrophic scar:

• **Induration:** raised above the surface but limited to the scar.

• **Course:** within months, it may regress.

• **Sites:** Common in shoulder & presternal area.

4. Keloid:

• **Def.:** overactivity of the healing process → excessive scar tissue.

• **Induration:** raised above the surface & extends beyond the scar.

• **Course:** Progressive even after 6 months.

• **Sites:** Ear lobules, shoulder, presternal area.

• **Incidence:** Black persons are more prone to keloid formation and there is a familial predisposition.

• **TTT of hypertrophic scars & keloids:**

a. Continuous pressure by silicone gel sheets.

b. Intralesional corticosteroids.

c. Surgical excision: Recurrence rate after simple excision may reach 80%. To minimize recurrence intramarginal excision of the scar + intraoperative injection of steroids are recommended.

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5. Contracture: pathological shortening of scar tissue → deformity if the scar overlies a joint. Proper positioning of the joint can minimize the deformity.

6. SSI.

Chronic wound

- Failed to undergrow the usual sequence of healing within reasonable time due to factor(s) affecting healing.
- They are arrested at the inflammatory & proliferative phases of healing with granulation tissue overgrowth, minimal contraction & collagen formation.



Management:

general & local factor management.

• **Examples:**

- Leg ulcers [venous (commonest), ischemic, neuropathic, neoplastic]
- Diabetic foot ulcers.
- Pressure sores (bed sores or decubitus ulcers):



Type of pt:

usually bed ridden pts due to high pressure applied on the skin.

• **RFs:** elderly pts, poor nutrition, anemia, denervation, paraplegia, ICU pts, immobilization & skin soiling by stools or urine.

• **Common sites:** ischium, greater trochanter, sacrum, heel & malleoli.



Stages:

1. Erythema.
2. Partial thickness skin loss.
3. Full thickness skin loss.
4. Further tissue destruction: muscles, bone & joints.



Prevention & TTT:

1. Correction of general factors as malnutrition.
2. Skillful nursing care as turning in bed.
3. Assure dry skin.
4. Protect pressure areas by cushions or foam.
5. Air mattresses.
6. Regular surgical debridement & the use of VAC.
7. Rotation flaps, myocutaneous or fascio-cutaneous flaps.

Accident & Emergency Surgery

Mechanism of injury:

1. Penetrating injuries:

- Low velocity injuries caused by
 - a. Knives, spikes of glass & other sharp objects.
 - b. Bullets fired from pistols (slow velocity).
- High velocity injuries caused by firearms caused by rifles.

2. Blunt injuries:

 e.g RTA, fall from a height or direct blows.

Causes of trauma mortality:

1. Immediate deaths:

- Occur within few mins after the accident → little to be done for the victims.
- Examples: major trauma to the brain or upper spinal cord, injuries to the heart or major vessel or rupture of the major airway.

2. Early deaths:

- Occur within hours after accident & can be prevented by rapid, proper management.
- It includes IC hge, massive intra-abdominal or intra-thoracic injuries or major fractures.

3. Late deaths:

 occur some weeks after the injury usually due to sepsis or MOF.

Organized trauma care:

- Victims of major trauma are best treated by a well-organized & trained trauma team.
- Accident & emergency departments should have an equipped resuscitation area to receive major trauma victims.
- In mass casualty accidents, the concept of triage is important. Triage means sorting of pts, i.e., their ranking according to both their clinical need & the available resources to provide ttt. It may take 2 forms:
 - a. If the number of casualties does not exceed the facilities all critically injured are treated.
 - b. If the number of casualties exceeds facilities, then the critically injured most likely to survive are treated 1st.
- The American College of Surgeons developed the Advanced Trauma Life Support (ATLS) accepted protocol for the management of major trauma victims. ATLS protocol has three elements
 1. 1st survey resuscitation.
 2. 2nd survey.
 3. Definitive care of individual injuries.
- The 1st survey & resuscitation should start at the site of accident by trained ambulance personnel.



Introduction

1-44

Trauma is the leading cause of mortality for individuals of age 1-44 years.

2

For every trauma, 2 people suffer permanent disabilities.

3rd

It's the 3rd cause of death in all ages.

1st survey Objective:

- to identify & treat any immediately life threatening condition.

2nd survey Objectives:

- Full examination.
- Complete history.
- Integration of all available data.
- Formulation of a management plan.

[1] Primary survey / Resuscitation

Steps

- Airway & cervical spine control.
- Breathing.
- Circulation with hge control.
- Disability (neurological assessment).
- Exposure & Environment.



- Tracheostomy is rarely needed in the emergency room management.

- Orotracheal intubation allows the use of a large tube.
- Nasotracheal intubation is safer if the cervical spine appears fractured.

[A] Airway:

- In general, if the pt is capable of unstrained speech, his airway is patent.
- All pts receive supplemental O₂ by mask upon arrival.

• Clear the airway:

1. Vomit, blood or foreign material should be removed manually (finger sweep) or with a rigid sucker.
2. This is followed by chin lift or jaw thrust.

• protect the airway:

- A. An oropharyngeal or nasopharyngeal tube prevents the tongue from falling back & occluding the airway in an unconscious pt.
- B. Tracheal intubation is indicated with:
 1. Apnea
 2. Risk of aspiration
 3. Impending or actual airway compromise (inhalation injuries, maxillofacial trauma)
 4. Closed head injuries (allow hyperventilation to ↓↓ ICT).
- C. Cricothyroidotomy is done either by making a cut & inserting a tube or by the percutaneous insertion of a wide-bore needle. It's not suitable for children.

• Cervical spine control:

- A. The cervical spine should be considered unstable in the following conditions:
 1. Clinical ex. reveals bony abnormalities or cervical tenderness.
 2. Multisystem trauma.
 3. Blunt injury above the clavicle.
 4. An altered level of consciousness from trauma or from drug alcohol intake.
 5. Maxillofacial trauma.
- B. Cervical spine immobilization is done by using a backboard & a rigid collar.
- C. If a collar is not available, manual in-line immobilization is necessary.
- D. Radiological evaluation [at least, 3 cervical spine views (lateral, AP & odontoid)] is done later after stabilization of vital systems.

[B] Breathing:

• Assessment:

1. Inspection for chest movement, RR, cyanosis, jugular venous distention, open chest wounds, asymmetric expansion & the use of accessory ms of respiration.
2. Palpation for SC emphysema & flail segments.
3. Percussion for hyperresonance or dullness.
4. Auscultation for upper airway sounds (stridor, wheezing or gurgling) & for lower airway sounds over the lung fields.

• Life threatening thoracic conditions: (TOM FC)

1. Tension pneumothorax. TTT: needle decompression followed by intercostal chest tube.

2. Open pneumothorax. TTT: occlusive dressing fixed at 3 sides only followed by chest tube.
3. Massive haemothorax. TTT: chest tube then thoracotomy later if bleeding continues.
4. Flail chest caused by fractures of adjacent ribs & commonly ass. with lung contusion. TTT: intubation & PPV.
5. Cardiac tamponade. TTT: needle pericardiocentesis followed later by operative pericardiotomy & control of the source of bleeding.

[C] Circulation:

• Shock:

1. Hemorrhagic (commonest): Tension pneumothorax ↓↓ VR & worsens it.
2. Cardiogenic: Tamponade & myocardial trauma.
3. Neurogenic: Spinal cord injury.

• Action:

1. Control of hge by direct pressure. Sites for hidden hge are intra-abd., intra-thoracic & fracture of pelvis & femur.
2. Insert 2 large calibre (16 gauge) peripheral I.V lines ± a central line.
3. Bl. samples are sent for typing, cross-matching, HB, Hct & bl. chemistry.
4. Ringer's lactate solution is infused as a start. Vol. of crystalloid needed = 3 x estimated bl. loss.
5. When cross matched bl. is available, it's infused immediately.
6. I.V fluids & bl. are given at a rate that ensures an optimum urine output of 0.5-1 ml/kg/hr for adults.

[D] Disability: (Brief neurological assessment)

• Common causes of neurological deficits related to trauma:

1. Head injury.
2. Hypoxia.
3. Shock.
4. Alcohol or drug abuse.

• AVPU Evaluation: according to best response:

- Alert & interactive.
- Vocal stimuli elicit a response
- Painful stimuli are needed to produce a response
- Unresponsive.

[E] Exposure & Environment:

1. Remove all clothes using sharp large scissors.
2. Keep the victim & place warm to avoid hypothermia
3. Insert urethral catheter (Foley's) to monitor urine output.
(Contraindicated if there is bl. at the urethral meatus which indicates urethral injury)
4. Insert nasogastric tube (Ryle's) to decompress the stomach & prevent vomiting & aspiration.
5. Radiological ex.:
 - For blunt trauma: cervical spine, AP chest & AP pelvis X-rays are mandatory.
 - For penetrating trauma: AP chest & X-ray of trauma site.
 - After resuscitation, other X-rays or CT scans are performed if indicated.
6. Proper history taking: (AMPLE).

AMPLE

Allergies
Medications
Past medical history
Last meal time
Events of injury

[2] Secondary survey

- It includes ex. of:

1. Head & neck for hematoma, fractures & pupils.

2. Face & spine [by 4 persons who turn the pt's body in one piece (log rolling) to avoid injury of the spinal cord if there is an unstable spine fracture].

3. Chest & abdomen.

4. Perineum including PR in all pts & PV in females.

5. Limbs: for fractures & soft tissue injuries including vs, nerves & tendons.

6. Nervous system:

- Pupils for size, equality & reaction to lights.
- GCS
- Cranial nerves.
- Sensation & motor activity in the limbs.
- CT scan of head if there is suspicion of intra-cranial injuries.

[3] Definitive ttt

Of individual injuries: according to its site (see before).



- A more detailed assessment using GCS is performed during the 2nd survey.
- In analyzing the causes of death in major trauma, 3 interrelated factors (hypothermia, coagulopathy & metabolic acidosis) make a vicious circle → potentially fatal outcome.
- Hypothermia → slowing of the clotting factors → Coagulopathy → ↓ tissue perfusion → anaerobic metabolism → Metabolic acidosis. So warming of the pt by blankets or warm IV fluids is essential.
- 5 causes of upper airway obstruction: tongue, blood, loose teeth or denture, vomitus & soft tissue edema.
- 5 main features of respiratory distress:
 1. Tachypnea &/or dyspnea.
 2. Use of accessory muscles of respiration.
 3. Difficult speaking.
 4. Low O₂ sat. at bedside oximetry e.g. 80%.
 5. Agitation or confusion.
- 5 areas of potentially severe bleeding :
 1. Chest
 2. Abdomen
 3. Pelvis
 4. Long bone fractures
 5. External bleeding
- At end of major survey , detect or exclude the following 5 serious problems:
 1. Airway obstruction.
 2. Massive haemothorax.
 3. Flail chest.
 4. Cardiac tamponade.
 5. Pneumothorax, open or closed.

Fluid, Electrolytes & Acid-Base Balance

Diagnosis of Imbalances

Symptoms

e.g., vomiting, diarrhea, or excessive sweating.

Signs

- Vital signs, pulse & BP.
- Urine output & Tissue turgor.
- Edema & crepitations.

Labs

- CVP & pulmonary artery wedge pressure (PAWP).
- HT & HB, Serum Na & K.
- ABGs to obtain Blood pH, PO₂ and PCO₂ & HCO₃.

CO-OXIMETRY		
THb	13.5	g/dL
FO ₂ Hb	96.7	%
FCOHb	2.3	%
FMetHb	0.3	%
FHb	0.7	%
OXYGEN STATUS 37.0 °C		
SO ₂	18.3	mL/dL
PO ₂ /F _{IO₂}	4.55	mmHg/%
PO ₂ (a)	19.8	mL/dL
CORRECTED 39.0 °C		
pH(T)	7.382	
PCO ₂ (T)	40.2	mmHg
PO ₂ (T)	467.5	mmHg
PO ₂ (a)(T)	199.8	mmHg
PO ₂ (a/A)(T)	0.70	
ELECTROLYTES		
Na ⁺	131.1	mmol/L
K ⁺	4.85	mmol/L
Ca ⁺⁺	4.5	mg/dL
Cl ⁻	104	mmol/L
METABOLITES		

Body Water

Total body water is 45-75% of body weight depending on fat content.

2/3 of the water is intracellular while the other 1/3 is extracellular.

ECF is subdivided into the interstitial compartment & intravascular compartment.

Water distributes freely & its source is either exogenous or endogenous.

Water loss is daily:

- Lungs (400ml)
- Skin (600-1000ml)
- Feces (60-150ml)
- Urine (1500ml)

Water Imbalance

	Water depletion (Pure dehydration)	Water excess (water intoxication)
Causes	1. ↓↓ water intake. "Lack of availability, Difficulty to swallow, Inability to swallow in coma" 2. DI. 3. ↑↑ output e.g. in fever & osmotic diuresis	1. Over-infusion of 5% glucose solution to post-operative patients. 2. Colorectal washouts with plain water. 3. TURP \$ (transurethral prostate resection) from irrigation fluid. TTT: Replace by glycine.
C/P	1. Intense thirst & weakness 2. ↓↓ Tissue turgor. 3. Oliguria with ↑↑ specific gravity.	Moderate cases: 1. ↑↑ Urine volume. 2. ↑↑ Body weight. Pitting edema usually does not develop. 3. ↓↓ Serum Na concentration & falling HT value Severe cases: 4. Rarely, swelling of brain cells → drowsiness, weakness&convulsions&coma (water intoxication) 5. Nausea, Clear fluid vomiting.

	Water depletion (Pure dehydration)	Water excess (water intoxication)
TTT	Na-free water acc. to the estimated deficit. If water depletion is severe, at least 1/2 of the estimated deficit should be replaced in the initial 12 hrs of ttt. 5% G is given IV.	Mild cases : Restriction of water intake. Severe cases: 1. Forced diuresis with Mannitol. 2. In severe cases IV administration of small amounts of 5% NaCl solution.

Sodium Imbalance

	Hyponatremia	Hypernatremia
Causes	1. Abnormal GIT losses. "Suction, vomiting and diarrhea mostly in small bowel obst. 2. Loss of ECF, either externally (burns, marked sweating) or internally as a 3rd space* (peritonitis, ascites or ileus). 3. Excessive urine Na loss e.g. (diuretics). 4. Blood loss. 5. Restricted dietary intake. 6. Adrenocortical insufficiency.	1. Giving excessive amount of 0.9% saline solution IV during the early postoperative period. 2. Hyperaldosteronism: 1ry (Conn's \$) or 2ry to liver cirrhosis. 3. Cushing's \$.
C/P	1. Eyes are sunken. In infants the anterior fontanel is depressed. 2. The tongue is coated & dry, Mild thirst. 3. Peripheral veins are collapsed. 4. Hypovolemia, with low CVP → tachycardia, orthostatic hypotension and shock with scanty, dark urine of high specific gravity. 5. There is hemoconcentration with normal or slightly reduced serum Na..	1. Slight puffiness offace is the only early sign. 2. Edema, the only reliable clinical sign. 3. Weight gain. 4. HTN. 5. Serum Na concentration is usually normal.
TTT	1. Restoration of ECF by appropriate amount of Na-containing fluids such as normal saline (NaCl 0.9%) or Ringer's lactate. 2. Blood losses should be replaced.	1. Na restriction. 2. Diuretics carefully.

*ECF collection not functionally available to normal mechanisms maintaining fluid & electrolyte imbalance.

Sodium
• Main extracellular cation that plays the main role in maintaining blood volume. • Adrenal corticoids, mainly aldosterone, control reabsorption of sodium in renal tubules. • Excretion is reduced after trauma & surgery for 48 hours, due to increased adrenocortical activity.

Potassium
• The major Intracellular cation. • Fruit, Milk, Honey are rich in it. • Excreted mainly in urine, very small quantity in feces & smaller quantity in sweat. • Augmented excretion follows surgery & trauma varying directly with tissue damage. It is greater in the 1st 24 hours & lasts ≈ 3-4 days. • Unless the patient is severely depleted of "K" at the time of operation, ↓↓ K may not reveal itself for 2 days as the reserves are great.

Calcium
• Serum Ca (8. -10.5 mg/dl) is found in ionized & non-ionized (albumin bound) forms. • Alkalosis decreases the ionized form & may lead to tetany. • It is controlled by vitamin D, PTH, Calcitonin, kidney & small bowel state.

Potassium Imbalance

	Hypokalemia	Hyperkalemia
Causes	1. Vomiting as in gastric outlet obstruction, e.g. pyloric obstruction. 2. External GIT fistulae. 3. Diarrhea especially when severe. "Cholera, Ulcerative colitis" 4. Certain types of diuretics as frusemide. 5. Alkalosis & Hyperaldosteronism.	1. Renal failure. Made worse by Na or Ca depletion or Tissue destruction. 2. Acidosis → K shift to ECF. 3. Diabetic pts. "Insulin → ↓↓ K".
Consequences	1. Raises membrane excitation potentials, making nerves & muscles less excitable. 2. Risk of supraventricular arrhythmias. 3. Increases the susceptibility of hepatic coma in pt with liver disease.	1. Stimulation of aldosterone secretion (K⁺ level 5mmol/L). 2. Decreases membrane excitation potentials, making cells more excitable.
C/P	1. Most patients are asymptomatic. 2. Malaise & weakness. The speech is slow 3. Paralytic ileus & distension may be seen. 4. Muscular paresis appears only with extreme depletion. Respiratory paresis → inadequate ventilation & atelectasis. 5. ECG reveals prolonged QT interval, depression of the ST segment, and a lowering or inversion of the T wave.	If the serum potassium exceeds 7mmol/L, intracardiac conduction is slowed and arrhythmia, bradycardia and hypotension may be followed by cardiac arrest. The ECG is a sensitive investigation for hyperkalemia. It shows a wide QRS complex and peaked T waves.
TTT	• The required quantity of K is added to the infusion & distributed all over the day. $[(4.5 - \text{Serum K concentration}) \times 100]$ • K should never be injected as a bolus. • Safe rules for giving K are: 1. Urine output at least 40 ml/hr. 2. Not more than 40 mmol added to 1 liter. 3. No faster than 40 mmol/hour.	1. IV Ca gluconate as Ca antagonizes K. 2. IV NaHCO₃. 3. Dextrose and insulin infusion. 4. If these measures fail, they should be followed by giving exchange resins "Na polystyrene sulfonate in 70% sorbitol" 5. If all the previous measures fail to lower the serum K, dialysis should be started.

× Acidosis → Hyperkalemia.

× Alkalosis → Hypokalemia.

Acid- Base regulation: (7.4 ± 0.04)

- Renal regulation by HCO₃ reabsorption & H⁺ excretion.
- Respiratory regulation by rate of CO₂ elimination.
- Buffers: Most importantly Bicarbonate : Carbonic acid=20:1.



	Metabolic Acidosis	Metabolic Alkalosis
Causes	1. Organic acid production: - DKA - Lactic acidosis due to sepsis, shock. 2. HCO_3^- loss: - RF (Acute, Chronic). - Diarrhea, pancreatic fistula, small intest. fistula, ureterosigmoidostomy	1. H^+ loss. "Vomiting, Gastric Suction" 2. HCO_3^- retention: NaHCO_3^- retention. - Milk alkali $\\$. 3. H^+ movement into cells with $\downarrow\downarrow$ K while K moves out to preserve electroneutrality.
Compensation	- Respiratory stimulation $\rightarrow \text{CO}_2$ washout. - Renal excretion of acid $\uparrow\uparrow$.	- Respiratory inhibition $\uparrow\uparrow \text{PCO}_2$ but limited by PO_2 reaching 50mmHg. "Hypoxic Drive"
C/P	$\uparrow\uparrow$ Rate & Depth of breathing. "Kussmaul's Respiration"	- Cheyne-Stokes respiration. - Tetany occasionally occurs.
TTT	1. Mild to Moderate: Treat the cause e.g. Restore tissue perfusion. 2. Severe: "pH 7.3 or serum $\text{HCO}_3^- < 15$ mEq/L" IV Bicarbonate (Amount required = Body Wt x 0.3 x Base Deficit).	1. Cl^- replacement in gastric juice loss. 2. Mild Cases: Saline $\pm$ IV K depending on the presence of $\downarrow\downarrow$ K. Kidney retains Cl^- & secretes Na with excess HCO_3^- . 3. Severe non responding cases: IV NH_4Cl or HCl very slowly. 4. Tetany: Slow IV 10 ml Ca Gluconate.

	Respiratory Acidosis	Respiratory Alkalosis
Causes	1. Hypoventilation. "Opiates, Anesthetics, CNS lesions" 2. Respiratory muscles or chest wall disorders: - Myasthenia, Poliomyelitis. - Morbid Obesity. - Flail Chest. 3. Other ventilation disorders. "COPD, Pulmonary Edema"	Hyperventilation: - Hysteria. - Hyperpyrexia. - Mechanical Ventilation.
Compensation	Renal compensatory mechanisms take time to $\uparrow\uparrow$ Serum HCO_3^- levels in chronic cases.	$\uparrow\uparrow$ Renal HCO_3^- excretion "Usually inadequate"
C/P	- Restlessness, Cyanosis. - HTN, Tachycardia immediately postoperatively.	- Usually short lived, Well tolerated. - Paraesthesia in the extremities, Carpopedal spasm due to $\downarrow\downarrow$ Ionized Ca. - Respiratory arrest in severe cases.
TTT	Mechanical Ventilation is usually required.	1. In hysteria, Breathing into a paper bag. 2. In other cases, Adding small amounts of CO_2 to the inspired gas mixture.

Hemorrhage

Hemorrhage may be classified according to:

[1] Site:

1. External: Bleeding is visible as it occurs through the skin as in wounds or from a body orifice as in epistaxis or hematemesis.
2. Internal:
 - a. In body cavities e.g. hemoperitoneum & hemothorax.
 - b. Interstitial e.g. fracture hematoma

[2] Type of disrupted vessel:

1. Arterial: The blood is bright red in color & comes in pulsatile jets.
2. Venous: The blood is dark red in color and comes as a steady flow.
3. Capillary: Bleeding occurs as diffuse ooze of bright red blood.

[3] Timing in relation to the onset of trauma:

1. 1st hge occurs at the time of trauma.
2. Reactionary hge: occurs some hours after trauma (within 24 hrs) due to slipped ligature or dislodged clot as BP rises.
3. 2nd hge: occurs one to two weeks after trauma due to infection eroding a vessel wall. It can be fatal if a large blood vessel is involved.

[4] Etiology: Hemorrhage may be:

1. Traumatic «Accidental, Surgical, Interventional (PTC)»
2. Pathological:
 - Atherosclerotic (ruptured aortic aneurysm).
 - Inflammatory (bleeding peptic ulcer).
 - Neoplastic (hematuria in renal cancer).
3. Bleeding diathesis: ↑ amount of traumatic & pathological bleeding or cause spontaneous bleeding.

Parameter	Class I	Class II	Class III	Class IV
Blood loss	Up to 15% (0.75 L)	15-30% (0.75-1.5L)	30-40% (1.5-2 L)	> 40% (2 L)
Mental status	Normal to anxious	Anxious to restless	Aggressive to drowsy	Drowsy to unconscious
Skin	N	Pale cold	Paler colder	Pale very cold
Capillary refill	N	>2 secs	>2 secs	>2 secs undetectable
Pulse/min.	<100	100-120	120-140	>140
Systolic BP	N	N (Supine)	Low	Low
Diastolic BP	N	High	Low	Low
Pulse pressure	N	Low	Low	Low
RR	14-20	20-30	30-35	>35
Urine output	>30 ml/hr	20-30 ml/hr	10-20 ml/hr	0-10 ml/hr

[5] Hge can be classified into 4 classes:

Physiological response, C/P, management: See hypovolemic shock.

Estimating blood loss: This is obtained from:

1. Clinical data.
2. Types of injury e.g. fracture of pelvis (2000 - 3000 ml).
3. Blood loss at operation is the sum of the amount in the suction reservoir and the amount mopped by the swabs (Swab weight after – before operation) x (1.5-2) acc. to the operation magnitude.

Investigations: A blood sample is obtained for:

1. CBC, including Hct.
2. Coagulation profile.
3. Cross matching.

Excessive Operative & Postoperative bleeding

Causes:

1. Inadequate surgical hemostasis (commonest cause)
2. Hemorrhagic diathesis not detected preoperatively as coagulation profile could be normal even with the deficient factor level down by 20%.
3. Acquired intraoperative coagulopathy e.g. DIC & massive blood transfusion.

Management:

1. Replace amount of blood loss.
2. PT, PTT & platelet count.
3. Fresh Frozen Plasma (FFP) 3-4 units initially & Platelet transfusion if needed.
4. Proper surgical hemostasis, correct hypothermia & acidosis.

Surgical hemostasis

During surgery the bleeding is controlled by one of the following methods:

1. Ligation.
2. Under-running suture.
3. Electrocautery (diathermy): to stop bleeding from small blood vessels, either Monopolar or Bipolar.
4. Clips Laser.
5. Repair of injured large vessels see arterial.



- 1st hemostasis is achieved by VC, platelet plug & tamponade by surrounding tissue tension
- 2nd hemostasis is achieved by coagulation.

Blood transfusion

Blood components:

1. **Packed red cells:** The transfusion of packed red cells is very useful in anemic pts, in the elderly and in cardiac pts as it improves the oxygenation ability without overloading the circulation.
2. **Fresh plasma:** It is rich in platelets & coagulation factors.
3. **Fresh frozen plasma:** Plasma removed from fresh blood is rapidly frozen and stored at - 40°C. It is a good source of all the coagulation factors. It's useful with hemophilia & liver failure.
4. **Platelet concentrates:** should be freshly prepared as half life of platelets is very short. It's useful in thrombocytopenia.
5. **Cryoprecipitate:** prepared from FFB & is very rich in factor VIII & fibrinogen. It is stored at -40°C.

Indications for transfusion of blood & its products:

	Indication	Precautions	Storage
Whole blood	Class III & IV hge.	ABO & Rh incompatibility	21 days
Red cell concentrates	Severe anemia	ABO & Rh	21 days
Fresh frozen plasma	1. Bleeding due to non-specified coagulation factor deficiency. 2. Coumarin overdose.	ABO	One year at -40°C
Platelets concentrates	1 st or 2 nd thrombocytopenia	ABO	24-72 hrs
Cryoprecipitate of factor VIII & fibrinogen	Bleeding with fibrinogen depletion		
Factor VIII	Hemophilia A		2 years
Factor IX	1. Hemophilia B. 2. Coumarin overdose.		
Albumin 50/0 or 200/0	Hypoalbuminemia		2 years



It is no longer recommended to transfuse fresh blood to correct coagulopathies. The specific deficient component should be used.

Complications of blood transfusion

- Though generally safe and simple, the procedure of bl. transfusion is not free of complications.
- The majority of these are simple, yet some are immediately fatal & others produce chronic serious illness as hepatitis & AIDS. Attention to strict rules of donation & administration of bl. greatly reduces the incidence of these complications.

I- In the donor:

1. Neurogenic shock.
2. Anemia if repeated amounts are taken.
3. Local thrombophlebitis in the veins.

II- In the recipient:

1. Immunological complications of bl. transfusion:

- Incompatible red cells → acute hemolytic reaction.
- Incompatible white cells → pyogenic reaction.
- Incompatible platelets → purpura.
- Reaction to a protein in the plasma → allergic reaction.

a. Hemolytic reactions

- Due to: presence of ABs in the recipient's blood against antigens in the donor's red cells.



Clinical Picture

- After the transfusion of < 50 ml: fever, chills, constricting pain in the chest, dyspnea & pain in the flanks, tachycardia, hypotension.
- In anesthetized pts: there will be sudden tachycardia, hypotension & bleeding tendency.
- A major hemolytic reaction → hemoglobinuria, jaundice & ARF.



Treatment

- Stop the transfusion immediately.
- Repeat typing & matching.
- Correct shock by infusion of crystalloid solution (Lactated Ringer) & IV corticosteroids.
- Monitor urine output.
- An osmotic diuretic as mannitol may be needed. Keep urine alkaline by IV bicarbonate to protect against ARF
- If ARF develops, it must be treated.

b. Pyrogenic reactions (The most common complication)



Clinical Picture

- Chills, fever, headache, nausea & vomiting.
- Due to: presence of recipient ABs against the donor's WBC's or bacterial contamination.



Treatment

Stop transfusion, Give aspirin or paracetamol IV.

c. Allergic reactions:



Clinical Picture

- Range from mild itching & urticaria to severe reaction with laryngeal edema & collapse.
- Due to: The recipient's response to allergens in the donor's blood.

Delayed hemolytic reaction after 5-10 days occurs in pts who have been immunized to a foreign antigen by previous transfusion or pregnancy. It presents by unexplained pyrexia or jaundice.



Treatment

antihistaminics & CS. If the reaction is severe, bl. transfusion should be stopped.

d. Post transfusion purpura.

2. CHF:

- Occurs in: elderly persons esp. if a large volume of blood is administered too rapidly.



Clinical Picture

See medical notes.



Prevention

It is recommended to transfuse packed red cells rather than whole blood to correct anemia in elderly persons.

3. Transmission of infection:

- Viral hepatitis (B or C): This is now the most feared complication. Whole blood or blood products can transmit the virus. It is now obligatory to test donor for hepatitis viruses.
- AIDS infection.
- Septicemia: Bacteria can survive, but they can't multiply significantly in refrigerated blood. However, if the bl. is allowed to warm, bacteria grow & G -ve endotoxins → septicemic shock
- Malaria by RBCs only.
- Syphilis: Can't survive at blood bank temperature for > 4 days.

4. Hyperkalemia :

- With prolonged storage of bl., there is progressive loss of K from erythrocytes into plasma.
- Transfusion of several units of aged bl. may produce cardiac arrhythmias or even arrest due to hyperkalemia.

5. Citrate intoxication:

- Excess citrate will bind to the recipient's Ca → hypocalcemia that augments the effects of hyperkalemia on the myocardium.
- Prevention: If > 2 units of blood are administered, it is important to administer 10 ml of 10% Ca gluconate for each 2 units of blood.

6. Air embolism.

7. Transfusion-related acute lung injury (TRALI)

- Due to incompatibility between donor's ABs & recipient granulocytes.



Clinical Picture

as ARDS.

8. Complications of massive blood transfusion:

- Def.: Transfusion of 2500 ml of blood at one time or 5000 ml or more over 24 hrs.
- a. Hypothermia: A special warming unit should be used to warm blood before transfusion as hypothermia → acidosis & arrest.
- b. Hyperkalemia.
- c. Hypocalcemia.
- d. Coagulation failure: As stored blood is poor in platelets, factor VIII & factor V. In these situations it is recommended to transfuse one unit of fresh frozen plasma & platelets for every unit of stored blood.
- e. Diminished O₂ carrying capacity of RBC's.

The physiological changes which occur during blood storage:

Items	0 day	7 th day	14 th day	21 st day
% of red cells viability	95%	90%	85%	75%
% of platelets viability	95%	0%	0%	0%
% of factors V & XIII	95%	30%	30%	30%
K content (mmol/L)	3.5	10	25	30

How to minimize the need for homologous blood transfusion?

1. Autologous blood transfusion:

- The pt who is going to have a major elective operation, can donate some units of his own bl. over several days. The bl. is kept in the refrigerator to be given back to him during surgery.

2. Preservation of the blood lost during surgery and its reinfusion to the patient:

- This needs a special apparatus (cell saver).

Shock

Pathophysiologic condition that leads to inadequate tissue perfusion through the microcirculation with impaired cellular metabolism.



Classification

1. Hypovolemic shock.
 2. Cardiogenic shock.
 3. Neurogenic shock.
 4. Anaphylactic shock.
 5. Septic shock.
 6. Endocrinal shock.
 7. Obstructive shock "Tension pneumothorax, Cardiac tamponade, Massive pulmonary embolism"
- Pt may have a combination of 2 or more of these types in some cases.
For example a trauma victim may have Hypovolemic shock from bl. loss
+ Obstructive shock from tamponade due to bleeding in the pericardium.

I - Hypovolemic Shock

**It is due to diminished blood volume,
which may occur 2ry to loss of:**

1. Blood: As in internal or external Hge.
2. Plasma: As in burns & acute pancreatitis.
3. Fluids: As in severe vomiting, diarrhea, intestinal obstruction & high output intestinal fistula.

Physiological response to Hge:

«The classical example of hypovolemic shock»

[1] Stopping the bleeding:

- Immediate VC & retraction of the intima with subsequent clotting. These mechanisms are more effective when the vessel is completely transected than when there is a side tear, and in traumatic than in pathological cases such as in peptic ulcer where fibrous tissue in its base hinder constriction.

[2] Maintaining effective circulatory volume & perfusion of critical tissues

(brain & heart) at the expense of less critical tissues (skin, skeletal muscle & splanchnic area) through:

A. Neural factors: ↓↓ Stimulation of baroreceptors «in the aortic arch & carotid sinus» & atrial stretch receptors → ↓↓ normal inhibitory vagal & glossopharyngeal discharge to the VMC → Sympathetic stimulation leading to:

1. Constriction of veins displaces blood from the capacitance side of circulation into the heart.
2. Constriction of arterioles raises the peripheral resistance. It involves mainly the arterioles of the skin, skeletal muscle, and splanchnic area.
3. Increased rate and strength of cardiac contraction.
4. Transcapillary refill: Constriction of arterioles causes a fall in capillary hydrostatic pressure and promotes movement of fluid from the interstitium into the capillaries → ↑↑ Blood Volume & ↓↓ Viscosity improving the effective circulatory volume.

B. Endocrine response: Is mediated through the following mechanisms:

1. Catecholamine: They increase the heart rate & myocardial contraction & cause constriction of the arterioles of the skin, kidney and viscera.
2. $\uparrow\uparrow$ ACTH, Cortisol, GH & Glucagon $\rightarrow$ to hyperglycemia which $\uparrow\uparrow$ ECF osmolarity & withdraws water from the ICF. This $\uparrow\uparrow$ effective blood volume.
3. The renin-angiotensin aldosterone system: Angiotensin II is a powerful vasoconstrictor and stimulates sodium and water retention by a direct action on the kidney as well as indirectly through aldosterone release.
4. Vasopressin (ADH): Absorbs water from the collecting tubules. At high concentrations, it causes VC.

- These mechanisms allow survival without therapy for losses up to 15% of the blood volume.
- Angiotensin mediated VC takes $\approx$ 20 minutes, Baroreceptor VC takes seconds.

[3] Microcirculatory changes:

1. Under the effect of catecholamines the precapillary sphincters constrict $\rightarrow$ Less blood enters $\rightarrow$ Sluggish capillary circulation.
2. Untreated hypovolemia thus leads to hypoxia. Under ischemic conditions, the body reacts by opening more capillaries. The results are:
 - Further slowing of capillary circulation. «Blood Sludging»
 - Spontaneous coagulation of blood in these capillaries. If extensive, it's called DIC.
3. A sluggish circulation & DIC aggravate tissue hypoxia and affect the function of capillary endothelium $\rightarrow$ Leakage of large protein molecules along with huge amounts of fluids into the interstitial space. Bleeding into 3rd space further reduces blood volume.

[4] Cellular Derangement:

1. Lack of O_2 forces the cells to rely on anaerobic glycolysis $\rightarrow$ lactic acid.
2. With persistent hypoxia, cellular functions start to deteriorate. « Na^+/K^+ Pump failure $\rightarrow$ Accumulation of intracellular water»
3. The end result is rupture of lysosomal and plasma membranes and cell death.
4. Vital organs are initially spared but eventually affected $\rightarrow$ Irreversible Shock.

[5] Acid-base imbalance: Shock is accompanied by metabolic acidosis which is caused by:

1. Accumulation of lactic acid.
2. Renal failure.

[6] Effect on individual organs: (see later)



Clinical Picture

«Depends on the amount & rate of Hge., Cardiovascular reserve»



Symptoms

1. Weakness and fainting especially when standing.
2. The patient feels cold and thirsty.



Signs

1. Mental status: From anxious to drowsy but the patient usually remains alert.

2. Pulse and blood pressure: With mild blood loss (< 500 ml), the pulse & blood pressure may remain normal. With more blood loss, tachycardia develops but blood pressure remains stable. With more progressive blood loss, progressive hypotension develops.

3. Pulse pressure: $\downarrow\downarrow \rightarrow$ thready pulse.

4. Respiratory rate: Tachypnea & air hunger due to chemoreceptor stimulation by hypoxemia & acidosis.

5. Temperature: Hypothermia occurs predisposing to coagulopathy.

6. Skin: Becomes pale, cold (vasoconstriction) & clammy with slow capillary refill & collapsed veins.

7. Oliguria: «Diminished renal perfusion»

- An atherosclerotic that has lost 500 ml blood may have hypotension whereas a soldier that has lost >1500 ml could have a normal systolic pressure early.

- If the patient is rapidly treated by adequate volume replacement, his vital signs will rapidly return to normal.
- If not, the following will occur:

a) The heart: Myocardial contractility is impaired by:

- o Direct myocardial depressants «Cachectin, Leukotrienes & PAF released in trauma, TNF from hypoperfused gut».
- o $\downarrow\downarrow$ Coronary perfusion.

b) Intestine: translocation of depressing substances (bacteria & endotoxins) from the ischemic gut into the circulation that may be a factor in the development of multiple organ system failure.

c) Stomach: true stress ulcers $\rightarrow$ Bleeding.

d) The liver: Ischemic Hepatic dysfunction is a frequent component of MOSE.

e) Renal affection: Prolonged hypotension ends in ATN which is part of the MOSE.

f) Pulmonary response: ARDS occur as part of MOSE.



Hypotension in supine position in a fit patient means a loss of at least 1500 ml of blood.

Irreversible shock

• Complete vascular collapse with hypotension unresponsive to volume or drug intervention leading to lethal central nervous system and cardiac dysfunction.

• It is related to:

1. The duration and volume of hemorrhage.
2. The age.
3. Pre-existing cardiovascular fitness of the patient.
4. The presence of massive trauma.

• Before the conclusion that irreversible shock has occurred, other causes of failure to respond to therapy should be resolved:

1. Continuing unsuspected blood loss into the chest or abdomen.

2. Inadequate volume replacement.
 3. Occult thoracic injuries including cardiac tamponade.
 4. Acute myocardial insufficiency.
- «From direct injury or prolonged coronary hypoperfusion»

Monitoring the severely shocked pt:

The aim is to check the adequacy of replacement:

1. **Clinical parameters:** Pulse, BP, state of filling of veins, capillary perfusion & urinary output/hr ($N=0.5-1$ ml/kg/hr).
2. **CVP:** A cannula is placed in the rt atrium via the median-cubital vein, the subclavian vein or the IJV. The normal pressure is 5-10 cmH₂O. Assuming that cardiac function is normal a high pressure indicates overtransfusion & the possibility of pulmonary edema, while a low pressure indicates hypovolemia.
3. **PAWP:** By Swan-Ganz catheter.
4. **ECG.**
5. **Temperature:** A simple non-invasive assesment of COP & peripheral perfusion is measuring the difference between the peripheral & core rectal temperature (Shouldn't be $>2^{\circ}\text{C}$).
6. **ABG:**
 - o PO₂ is normally between 80-100 mmHg.
 - o PCO₂ is normally between 35-45 mmHg.

7. Repeated Hct & Hb.



Treatment

of hypovolemic shock:

[1] **Stop the Hge** (if the cause of shock was hge, the classic example of hypovolemic shock):

[A] First-aid ttt:

1. By packing, pressure, & position.
2. A skin wound is covered by a dressing (any clean material), & pressure is applied manually, by a sphygmomanometer cuff or by a bandage.
3. Elevation of the limb above the heart level stops venous & decreases arterial bleeding.
 - Tourniquets are contraindicated due to complications unless the limb is going to be amputated.
 - Pneumatic antishock garment (PASG) can tamponade LL, pelvic, abdominal bleeding. Balloon tamponade is used in esophageal varices.

[B] Operation includes:

1. 1^{ry} Hge: It is treated by controlling the bleeding point either by ligature or diathermy coagulation.
2. Reactionary Hge: Open the wound & deal with the bleeding vessel as above.
3. 2^{ry} Hge:
 - The wound is packed with antibiotics and sterile dressing.
 - Antibiotics are given systemically.
 - If the bleeding is not controlled in this way, reopen the wound and drain the infection. The vessel is then exposed & the wound is ligated.

[2] Restore the blood volume:

«2 large gauge catheters are inserted into appropriate veins. Blood is drawn for typing & matching»

Class II (15-30% Blood Loss): «750-1000 ml»

- The replacement solution is lactated Ringer's.
- The amount is 3 times the estimated deficit ($\approx 3L$) as crystalloids diffuse out of the capillaries.
- Administration: 2L are given as a bolus and the response is monitored. If there is definite improvement, the remaining liter is given more slowly (Hct <30 requires blood transfusion).
- Moderate improvement indicates Inadequate replacement, Continuing bleeding, Myocardial insufficiency.

Class III and IV (30-40% & >40%):

- The management is as for class II but these patients need blood transfusion.
- The initial volume of transfused blood is that of the estimated deficit (1500 - 2000 ml).
- Clean blood in the thorax or abdomen can be aspirated, anticoagulated and then transfused back into the patient (auto-transfusion) if a cell-saver is available.
- In the absence of whole blood, many substances have been proposed as human plasma, albumin solution, dextran and artificial blood substitutes.
- Continue till Hct becomes 30%, Urine output 50 ml/hr, CVP $\uparrow\uparrow$ to upper $\frac{1}{2}$ of the normal range.

[3] Positioning: Elevate both legs while maintaining the trunk & the remainder of the pt in supine position.

[4] Pulmonary support:

- O₂ is administered through a facemask, nasal catheter or an endotracheal tube. Initially, high conc. is given then adjusted according to ABG results.

[5] Analgesia:

- Morphia 3-5 mg (poorly absorbed from SC sites). Contraindicated in head injury & in cases of respiratory & liver insufficiency.

[6] Inotropics: «2nd line of ttt»: Dopamine & Dobutamine are the most widely used; both improve myocardial contractility, while dopamine $\uparrow\uparrow$ RBF.

[7] Monitoring.

Volume replacement depends on the class of the Hge.

× Failure to improve & rising CVP:

- Tension Pneumothorax.
- Cardiac Tamponade.
- Cardiac Failure.

II - Septic Shock

+ Aetiology

• The incidence of G - ve sepsis is rising due to :

1. Development of resistant & virulent organisms.
2. Concentration of infected pts in critical care units.
3. More extensive operations in elderly & poor-risk pts.
4. Salvage of severely injured pts.
5. Pts who are immunosuppressed by organ transplantation, radiotherapy & chemotherapy.



This is the most serious type of shock & the most difficult to treat, having an overall mortality exceeding 25% & is exceeding 90% if with MOSF.

• **PFs:** Old age, DM, corticosteroids, chemotherapy, malignancy & AIDS.

• **CO:**

1. G -ve bacilli (commonest).
2. Staphylococci.
3. Candida.

• **Source of G -ve sepsis:**

1. Peritonitis caused by perforated viscus, gangrenous bowel or leaking anastomosis.
2. Cholangitis or genitourinary infections.
3. Infected central venous catheter.



Pathophysiology

A. Mediators cascade: Under the influence of endotoxins, macrophages & kupffer cells release cytokines (TNF, ILs, PAF, PGs, NO) that cause the following major problems:

1. Platelets & leucocytes adherence to the vascular endothelium →
 - Impairment of microvascular perfusion.
 - Local release of cytokines and free O₂ radicals that damage the vascular endothelium.
2. Damage of barrier function of the intestinal mucosa allowing intestinal pathogens into the circ..
3. Excessive production of NO by the vascular endothelium which is a potent smooth ms relaxant → ↓ peripheral resistance.

B. Microcirculation:

1. Under the effect of cytokines, arterial VD occurs & A-V shunts are opened. The results are:
 - ↓↓ peripheral resistance.
 - Capillaries are bypassed & O₂ delivery to the tissues is impaired.
2. DIC is the result of sluggish capillary circulation & PAF activation.
3. Vascular endothelial damage under the effect of cytokines is the dominant harmful feature of septic shock → Leakage of protein-rich fluid to the interstitial fluid → Edema.

C. Cellular derangement: (see hypovolemic shock):

It is to be noted that oxygen uptake & utilization by cells is also directly suppressed by the toxins.

D. Acid-Base imbalance. (see hypovolemic shock)

E. Individual systems & organs. (see hypovolemic shock).



Clinical Picture

	Hyperdynamic (warm)	Hypodynamic (cold)
	Diagnosis is difficult and a high index of suspicion is required to detect cases at this early stage.	If the previous stage is not treated efficiently, the pt will develop a picture similar to that of hypovolemic shock with reduced COP.
C/P	• Fever above 38°C & chills. • Pt is flushed with warm dry extremities • Restlessness & confusion. • Mild reduction in BP. • The COP is elevated. • Tachycardia & tachypnea. • Oliguria	• Cold clammy skin. • Systolic BP < 90 mmHg. • Tachycardia & tachypnea. • Oliguria. • MOSF starts at this stage
TTT	• Prompt & aggressive ttt of the pt at this stage can lead to survival.	• Efficient & early ttt of the pt at this stage, can convert him to the hyperdynamic stage. • Mortality rate of a septicemic pt with MOF exceeds 80%.



Diagnosis

Helped by:

1. PMNL with shift to the left.
2. High lactate level in blood.
3. Looking for a source of sepsis.
4. Repeated bl. culture at the peak of fever or culture from the septic focus to guide antibiotic ttt at a late stage.

Monitoring: See hypovolemic shock.



Treatment

(better in an ICU).

I. Support of different systems:

1. Cardiovascular support:

- Fluid replacement: Aim: CVP 10-12 cmH₂O, PAWP 12-15 mmHg
 - a. Prompt correction of fluid deficit is essential.
 - b. Ringer's lactate is usually used.
 - c. Huge quantities of fluids are often needed
 - d. The amount often exceeds 10L within a few hours.
 - e. Packed RBC's transfusion may be indicated in cases with low hematocrite.
- Medications: Inotropic drugs as dopamine & vasopressor drugs as norepinephrine to keep MAP >65 mmHg can be used in pts with persistent life threatening hypotension inspite of active measures of ttt. Epinephrine or Dobutamine are used in resistant cases.



Steroids are not indicated in the ttt of septic shock.

SIRS

- Septic shock is now considered to be a part of syndrome (SIRS).
- The cascade of events including mediators release and ending with translocation and bacteria are common with other members of syndrome which are :
 - Major trauma
 - Major burns
 - Acute pancreatitis
 - Neglected hypovolaemia

2. Respiratory support: Oxygen administration is essential for all types of shock. Usually 100% O₂ is administered as a start & is later adjusted acc. to the response. If the arterial O₂ is mildly ↓↓, O₂ by mask will be sufficient. ↓↓ its level < 60 mmHg calls for endotracheal intubation & MV.

3. Renal support: Adequate vol. replacement & dopamine improve RBF, hemodialysis in ARF.

II. Fighting infection:

«Huge abscess drainage, Resection of a gangrenous colon»

1. Immediate recognition & early eradication of the source of sepsis.

2. Antibiotics: The choice will depend on the possible suspected organisms. The most commonly used combination is cephalosporin, aminoglycosides & metronidazole. Modify acc. to C&S results.

III- Cardiogenic Shock

• Here there is inadequate bl. flow to vital organs due to inadequate COP, despite a normal bl. volume.



Aetiology

1. Acute MI (commonest cause).
2. Massive pulmonary embolism.
3. Cardiac tamponade.
4. Severe arrhythmia.
5. Improper ttt of hypovolemic shock may → cardiogenic shock.
6. Myocarditis, High spinal anesthesia (paralysis of sympathetic supply to the heart)



Clinical Picture

congested neck veins, ↑CVP, ↑SBP, ↑DBP, cold sweaty skin (compensatory VC) & metabolic acidosis



Treatment

1. O₂ Inotropics.
2. Vasodilators to reduce after load increase cardiac output and decrease myocardial work.
3. Early thrombolysis for MI & Potent Analgesics.
4. Control arrhythmias, Pericardiocentesis for tamponade.
5. Intraaortic balloon device.

IV - Neurogenic Shock

- There is paralysis of the vasomotor fibers → peripheral pooling of blood & inadequate VR. It may a
 1. Vasovagal attack:
 - It may follow hearing bad news, seeing unpleasant events or severe painful stimuli as blow to the testis.
 - There will be extensive VD in splanchnic area → ↓VR → ↓Bl. supply to vital areas & excessive vagal Stimulation of the heart

that causes bradycardia. Fainting assists VR → Recovery.

2. High transection of the spinal cord or complication of high spinal anesthesia, Deep General Anesthesia.



Treatment

- The pt should lie flat with elevation of the legs.
- I.V. crystalloid solution as Ringer's lactate.
- Vasopressors may be prescribed.

V- Anaphylactic Shock

- After penicillin, sera, anesthetics dextran → Ag-Ab reaction → Release of large amounts of histamine.
- The pt develops bronchospasm, laryngeal edema & RD. Massive VD occurs & there is hypotension.



Treatment

- IV crystalloid infusion.
- IV hydrocortisone.
- Antihistaminics.
- ETT with laryngeal edema, stridor.

VI- Endocrinal Shock

- This may occur in pts with Addison's disease or those receiving continuous cortisone therapy if they are subjected to any stressful situation «Infection, Surgery» due to failure of cortisol release.



Treatment

IV hydrocortisone, Saline infusion, TTT of predisposing factors.

VII-Obstructive Shock

- May occur in pt with:
 - tension pneumothorax.
 - cardiac tamponade.
 - Massive pul. embolism.
- It is due to reduced filling of the Rt side of heart. The characteristic findings are:
 - Low cardiac output.
 - High jugular venous pressure and CVP.



In neurogenic shock there is hypotension, normal pulse rate or bradycardia & warm dry skin. «Loss of sympathetic outflow»

Surgical Infections and Antibiotics

SSIs are infections of the **tissues, organs** or **spaces** exposed during the performance of an **invasive** surgical procedure. SSIs are classified into:

- Incisional: There are either superficial limited to the skin and subcutaneous tissues, or deep involving the musculoaponeurotic layers.
- Organs.
- Spaces: e.g., subphrenic, pelvic or interloop abscesses.

The development of SSIs is due either to general factors related to the patient or local factors related to the wound.

+ Aetiology

General host factors

- Old age.
- Obesity.
- Anaemia and malnutrition.
- Immunosuppression as in diabetes mellitus, uraemia and malignancy.
- Intake of drugs which suppress the immune system as corticosteroids, chemotherapy and immunosuppressive drugs.

Local factors

- Poor **skin** preparation.
- Defect in **sterilization** of the instruments or lack of adherence to theatre discipline.
- **Prolonged** surgical procedures.
- Improper surgical **technique**, e.g., leaving dead spaces, haematoma, too much use of diathermy or rough handling of tissues.
- Poor **blood** supply. Prolonged periods of hypotension and suture of tissues under tension predispose infection.
- **Nature** of the operation. This is the most important factor which determines SSIs, as most of these infections are endogenous due to microorganisms from the patient himself.

Surgical wounds are classified based on the resumed magnitude of the bacterial load at the time of surgery into

o Clean wounds (Class I):

These are elective non-traumatic wounds in which the gastrointestinal, urinary or respiratory tracts are not entered. Risk of SSI is less than **2%**.

o Clean contaminated (Class II):

Elective surgery into the gastrointestinal urinary or respiratory tracts with no significant spillage. Risk of infection is **2-5%**.

o Contaminated wounds (Class III):

- Open accidental wounds encountered within 4 hours.
- Gross **spillage** from the gastrointestinal tract.
- Incision through **inflamed**, non-purulent tissues Risk of infection is **10-20%**.

o Dirty wounds (Class IV):

- **Traumatic** wounds more than 4 hours.
- **Purulent** infection or necrotizing soft tissue infection.
- **Perforated viscus**: risk of infection is up to **40%**.
- Presence of **foreign bodies** or prosthetic material.



Clinical Picture

- Surgical site infection usually appears between the **fifth** and **tenth** days postoperatively but may sometimes appear earlier or later.
- Increase wound **pain** & postoperative **fever** are the earliest manifestations.
- Wound is **swollen** (sutures are dipping through the skin), tender and red.
- Fluctuant areas or crepitus are occasionally felt.
- **Discharge** from the wound may be seen.

Recommendations To Minimize Surgical Site Infections

The patient:

- **Correct** any predisposing factors as control of diabetes, stopping of smoking and correction of **nutritional deficiency**.
- Operations are avoided in patients with **active infections**, if possible.
- **Shaving or clipping** the hairs just before skin incision.
- **Skin preparation** with antiseptics.

The surgeon:

- Surgeon should have **short nails** and should scrub properly.
- Meticulous surgical **technique** by adequate haemostasis, gentle handling of tissue and **avoiding tight sutures** or leaving dead space.
- **Delayed primary** closure of heavily contaminated wounds.

Prophylactic antibiotics:

- Prophylactic antibiotics are indicated in clean contaminated or contaminated surgery, and in wounds where foreign material is implanted.

- Prophylactic antibiotics should be given **pre-operatively** and sometimes **during the operation** and in the **early post-operative period**.

The antibiotic is given **one hour before** surgery. A **booster** dose may be given in a **lengthy operation**. The administration of further doses depends on the degree of wound contamination. Prophylactic antibiotic administration **rarely exceeds one day**.

- **First generation cephalosporin** (cephazolin) is usually used but each hospital has an **infection control committee** that recommends an antibiotic according to its prevalent microorganisms.



- Infections deep to the deep fascia may be difficult to recognize. They are associated with systemic signs of infection.
- Clipping is preferred as shaving induces tiny abrasions that are prone to be colonized with bacteria.



Treatment

Liberal drainage:

The wound should be opened by removal of skin stitches.

- **Antibiotics** are used in invasive infections guided by culture and sensitivity tests.
- **Possible sources** of hospital acquired infection should be traced and corrected.

Cellulitis



Definition

Cellulitis is an **invasive non suppurative** infection of the loose connective tissue.



Aetiology

Causative Organism:

Mostly **streptococci**, or occasionally staphylococci into the superficial skin structures.

Mode of transmission:

The portal of entry may be trivial, e.g. a scratch or a prick.



Clinical Picture

- The affected area is red or **reddish, indurated**, hot and **painful**.
- It spreads rapidly and the advancing edge is **ill defined**.
- A moderate to high fever is always present.
- Lymphatic spread produces **red streaks** of lymphangitis and enlarged tender regional lymph nodes.



Treatment

- Antibiotics (**penicillin** group).
- Rest and elevation of the affected part.
- **Warm** packs.



Definition

This is a **rapidly spreading non-suppurative** inflammation of the **lymphatics** of the skin.

Causative organism:

A specific strain of **haemolytic streptococci**.

Mode of transmission:

Through a minute scratch or abrasion.



Clinical Picture

- Fever, malaise and **loss of appetite**.
- Locally the picture is similar to cellulitis, but there are some differences.
 - The skin is **rose-pink**.
 - The edge is **well-defined**, slightly **raised** and often shows **minute vesicles** just beyond the spreading margin.
 - There may be **islets of inflammation** beyond the spreading margin separated from the main area by apparently normal skin.



Complications

1. **Cavernous sinus thrombosis**: in Facial erysipelas.
2. **Septicaemia**.
3. **Elephantiasis** Recurrent erysipelas may block the lymphatics.



Treatment

Like cellulitis, treatment is by antibiotics, mainly **penicillins**. Commonly effective on intravenous administration.

Although polymorphonuclear leucocytes are abundant, there is no pus formation.

If no response to it has occurred within 48 hours, either an abscess has developed, or resistant organisms as staphylococci are involved.

Boil (furuncle)



Introduction

- This is a **staphylococcal** infection of a **hair follicle**.
- It can occur in any hairy area of the skin and is particularly common in the **face, neck and axilla**.



Clinical Picture

Clinically a boil forms a small **painful indurated** swelling which is hot, red and **very tender**.



Treatment

- **Antibiotics** effective against staphylococci.
- Warm **foments** facilitate necrosis of the overlying skin and escape of pus and slough.
- **Painting** the surrounding skin with an **antiseptic** to prevent infection of neighbouring hair follicles.
- Always **suspect diabetes** mellitus in patients who develop recurrent boils.

- 
- Boils are more common in diabetics and whenever there is lack of personal hygiene.
 - Early treatment may allow the boil to resolve without suppuration (Blind boil). More often, necrosis of the central part occurs and is discharged together with a small bead of pus.

Carbuncle

- This is **infective gangrene** of the **subcutaneous** tissues, usually secondary to infection by *Staphylococcus aureus*. It is common in **immunocompromised** patients as diabetics.
- The infection occurs mainly in the **face, nape** of the neck and the **back**.



Pathophysiology

Infection usually starts in a **hair follicle** and extends to the **subcutaneous** fat where **other hair follicles** get the infection. Multiple areas of **necrosis** and **thrombosis** of blood vessels occur. Patches of **skin** undergo **sloughing** and result in sinus formation.



Clinical Picture

There is usually severe toxemia. The carbuncle starts as a **painful induration** of the skin and subcutaneous tissues. The skin is red. As the swelling enlarges, its central part becomes soft but usually **no fluctuation** can be elicited. Multiple areas of skin thin out and separate forming **multiple sinuses**, through which small amount of thin pus trickles out.



Complications

- Local spread of infection leads to **cellulitis** and **lymphangitis**.
- **Septicaemia**.
- Carbuncles of the dangerous areas of the face can lead to **cavernous sinus thrombosis**.



Treatment

- **Antibiotics** effective against resistant staphylococci.
- **Culture and sensitivity** of the discharge.
- Control of **diabetes mellitus**, if present.
- Surgical **excision of sloughs** is commonly needed if there is no response to conservative treatment.

Hidradenitis suppurativa (suppurative hidradenitis)

Mixed staph. and streptococcal infection of the **apocrine** sweat glands, in the perineum or the axilla, produces multiple abscesses and pus-discharging sinuses. Infection is **difficult** to eradicate and the case usually progresses to **chronicity** with considerable fibrous tissue reaction.

In the perineum, hidradenitis suppurativa is often misdiagnosed as **multiple anal fistulae**.

Careful examination reveals the **absence of internal anal openings**.

Treatment is by **drainage** of abscesses followed by careful **hygiene**, painting with disinfectants and **antifungal applications** may be enough. Otherwise, **excision** of the apocrine sweat-bearing skin followed by skin **grafting** is needed.

Acute abscess

An abscess is a **localized suppurative** inflammation. It is caused by pyogenic organisms.

The commonest causative organisms are **staphylococci** that produce a coagulase enzyme which helps localize the acute inflammatory processes.



Pathogenesis

The organisms reach the tissues by

- **Direct access:** through wounds, scratches, and abrasions, or along natural passages such as lactiferous ducts.
- **Local extension:** from an adjacent focus, e.g. osteomyelitis of the jaw from an infected tooth.
- **Lymphatic spread:** Infection reaches the regional lymph nodes along lymphatics from a septic focus in their drainage area.
- **Blood spread:** Organisms gaining access into the circulation, as in bacteraemia or pyaemia, may lodge in distant tissues and cause abscesses e.g. pyaemic liver, and lung abscesses.



Pathophysiology

An abscess consists of three zones.

- **Central “zone of coagulative necrosis”:** This ultimately separates from surrounding tissues and forms a slough which becomes **liquefied** by the enzymes of dead leucocytes >> pus.
- **An intermediate zone of granulation tissue:** forms a protective layer against the spread of bacteria and their toxins.
- **A peripheral zone of acute inflammation:** fades gradually into healthy surrounding tissues.



Fate

- **Pointing and rupture:** is the commonest sequel. The abscess discharges its pus along the **plane of least resistance**.
- **Spread of infection:** either locally, by lymphatics, or by blood stream.

Chronicity: A dense fibrous tissue reaction forms around, leading to the formation of a mass with little inflammatory reaction around.

- This occurs when an abscess is treated by a prolonged course of antibiotics or if **inadequately drained**.



Clinical Picture



Symptoms

A **painful tender swelling**.



Signs

- The covering skin is **red**, and **oedematous**.
- The draining **lymph nodes** are usually enlarged and tender.
- A **systemic reaction** in the form of fever, malaise, headache, tachycardia and anorexia may be found.

The formation of pus is indicated by the following features

- Pain becomes **throbbing**.
- Fever becomes **hectic**.
- The covering skin shows **pitting oedema**.
- The inflammatory reaction becomes **localized**.
- There is shooting **leucocytosis with shift to the left**.



Treatment

Once pus forms, the only treatment should be free **drainage**.

- Incision and drainage is the standard method.
 1. The incision should be situated over the **pointing** part of the abscess and should be **dependent**; otherwise a **counter incision** over a dependent area is necessary.
 2. The **loculi** inside the abscess are **broken** to ensure adequate free drainage
 3. A **rubber drain** or a wick of gauze is left protruding from the abscess cavity.Adequate free drainage is usually enough for most abscesses. Antibiotics are required only in overwhelming infections or in immunocompromised patients.
- Ultrasound or CT-guided aspiration drainage is applicable for deep seated collections, e.g., an intraperitoneal abscess.



Fluctuation can usually be elicited in a subcutaneous abscess. This sign is absent or difficult to elicit in

- Deep abscesses (e.g. perinephric or gluteal abscess).
- Abscesses covered by strong fascia (e.g. parotid abscess).
- Breast abscesses as the normal breast tissue gives a false sense of fluctuation.

So, WE DO NOT WAIT FOR FLUCTUATION IN THESE AREAS AS IT IS A LATE SIGN) SPs Perinephric - Buttocks- Breast- Parotid-Pilon (distal Pulp space).

Acute Lymphangitis & Lymphadenitis Necrotizing fasciitis (Non-Clostridial necrotizing infection)



Introduction

This is an **invasive** infection that is usually caused by **mixed** microbial flora, microaerophilic streptococci, staphylococci, Gram-negative bacteria and anaerobes, especially **peptostreptococci**, and **bacteroides**.

Mode of transmission:

The organisms gain access through a **puncture wound**, **leg ulcer** or a **surgical** wound. Infection is more likely to occur in immunocompromised patients, e.g., diabetics and with malignant neoplasms.



Pathophysiology

The infectious process spreads **along the fascial planes** and results in infectious **thrombosis of its vessels**>>Superficial skin necrosis follows. It **resembles** ischaemic vascular or Clostridial **gangrene**.

Haemorrhagic bullae appear as the first sign of skin death. Fascial and subcutaneous fat necrosis involves an area wider than the skin.



Clinical Picture

- General:

The patient is **alert** and fully conscious. There are manifestations of **toxaemia** with fever and tachycardia.

- Local:

The skin shows **haemorrhagic bullae** and necrosis surrounded by **oedema** and inflammatory signs. **Crepitus** is occasionally present and the skin may be **anaesthetic**.



Investigations

- Gram stained **smears & cultures** show the mixed nature of the infection.
- **At surgery**, the finding of oedematous, **dull gray fascia** and subcutaneous tissue with **visible thrombi** in **penetrating vessels**, **confirms** the diagnosis.



Prevention

Adequate **debridement**, blood loss replacement & **antibiotics** for contaminated wounds are essential to prevent postoperative necrotizing fasciitis.



Treatment

• Surgical treatment:

Debridement, under anaesthesia, with removal of necrotic skin and fascia is essential. Muscles are usually healthy and the limb can be salvaged.

• Antibiotics:

Penicillin 20-40 million units/day intravenously together with **Gentamicin** (5 mg/kg/day) or **Amikacin** (15 mg/Kg/day) are essential to control the mixed infection.

Material is sent for smears & **culture** & sensitivity testing before treatment.

- **Blood transfusion, nutritional** support and control of **diabetes**.

Necrotizing fasciitis of the perineum or scrotum is known as **Fournier's gangrene**.

Tetanus



Definition

Tetanus is a **specific anaerobic** infection. leads to nervous irritability and tetanic muscular contractions.



Aetiology

Caused by the **neurotoxin of Clostridium tetani**.

Prevalence: The disease is becoming less prevalent thanks to the **compulsory immunization** in childhood.

Organism:

Clostridium tetani is a gram **positive** anaerobic bacillus with a terminal spore giving the characteristic **drumstick** appearance.

The organism is naturally present in the intestine of horses and the spores are present in manured soil and street dirt.

Mode of transmission:

The causative organism enters and flourishes in :

1. Hypoxic wounds: contaminated with **soil or faeces**. The tetanus-prone wound is usually a puncture wound or one containing devitalized tissue, a **foreign body**, or associated pyogenic organisms.

2. Umbilical stump: **Tetanus neonatorum** arises from infection of the umbilical stump by contaminated dressings or powders.



Pathophysiology

-The bacillus remains at the site of inoculation but its exotoxin reaches the central nervous system along the **blood** stream, the **motor nerves** or **both**.

- Once the toxin reaches the nervous system, it is at once fixed by the **motor cells** and cannot be detected in the blood or CSF.

Tetanus antitoxin can neutralize the toxin **only before it gets fixed** to the nervous tissue.

The toxin **increases excitability** of the motor cells of the medulla and spinal cord>> slightest stimuli produce violent spasms.

Death results from **exhaustion, hyperpyrexia, heart failure, asphyxia or pneumonia**.



Clinical Picture

Incubation period: Varies from 24 hours to **15 days**.

Tonic stage:

The first symptoms are usually **pain** and **tingling** in the area of injury, limitation of movements of the jaw (**lockjaw, trismus**) and spasm of the facial muscles (risus sardonicus).

These are followed by stiffness of the neck, difficulty in swallowing, and **laryngospasm**. Hesitancy in micturition due to sphincter spasm is also seen.

Clonic stage:

In more severe cases reflex **paroxysms** of violent muscular **contractions** become superimposed on the above mentioned tonic rigidity which are initiated by any form of stimulation such as **bright light**. During these spasms, the body may be **arched (opisthotonos)**. Spasms become more frequent and involve more muscle groups. As spasms of the intercostal muscles and diaphragm occur, longer periods of apnoea follow.

The **temperature** is normal or slightly elevated. Sweating is profuse.
Marked tachycardia is a grave sign.



Investigations

Polymorphonuclear **leucocytosis** may be present.



Differential Diagnosis

- Trismus due to local causes such as impacted wisdom tooth or temporomandibular arthritis. This can be excluded by careful local examination.
- Meningitis. The neck muscles are affected first and the CSF is turbid and contains leucocytes and organisms.
- Strychnine poisoning. Spasms start in the extremities and are entirely clonic with complete relaxation during the intervals.
- Tetany. The hands and feet are mainly affected. **Chvostek's** sign is positive and serum calcium is low.
- Rabies: There is history of a dog-bite. The spasms occur on seeing and drinking water.

The muscles of **deglutition and respiration** are mainly affected.



Prevention

- **Every child** should be actively **immunized** with tetanus toxoid, beginning with routine childhood immunization and continuing with **booster** injections every 7-10 years.
- **Previously immunized** individuals by three or more doses, the last **within 10 years** need only a **booster** dose (0.5 ml IM).
- **Not previously immunized** individuals should receive the following doses
 - o **Clean minor wounds** (tetanus is unlikely).
 - IM 0.5 ml of tetanus **toxoid** is given as the initial dose.
 - Two further injections are given at 4-week intervals.
 - o **Wounds with high tetanus risk.**
 - The initial **immunizing dose** (0.5 ml intramuscularly) of the tetanus toxoid is given together with **250** units intramuscularly of tetanus immune globulin (**TIG**) in a different syringe and at a different site, and consider the use of **antibiotics**. Plan to complete the toxoid series.



Treatment

Intensive treatment **should be started soon**, as respiratory paralysis may advance rapidly.

A. Neutralize toxin with TIG.

- The dose is **3000-6000** units intramuscularly, given preferably in the **proximal** portion of the wounded extremity (near the wound).
- **Repeated doses** may be needed since the half-life of the antibody is about 3 weeks and established tetanus often lasts longer.

B. Excise and debride the suspected wound under anesthesia.

C. The patient with respiratory problems may require **tracheostomy**, since mechanical ventilation, once it becomes necessary, must be continued for weeks.



• Barbiturates are used cautiously as they often cause cardiorespiratory failure.

• Diazepam may reduce the dose of barbiturates needed to control spasms. Muscle relaxants with mechanical ventilation is a better alternative to large doses of barbiturates.

D. Endo-tracheal intubation: once respiratory problems appear.

E. Aqueous penicillin G: **10-40 million units** a day, by intermittent **intravenous** bolus injection should be given to kill Clostridial organisms and prevent further release of toxin.

F. Nursing: The patient is **isolated** in a **dark** quiet room and protected from sudden stimuli, unnecessary movements and excitement.

Nutrition is maintained by a **nasogastric tube**.



Prognosis

- Mortality rate is **30-60%** in established cases with respiratory insufficiency.
- This rate is **inversely proportionate** to the length of the **incubation period** and directly proportionate to the severity of symptoms.
- One attack of tetanus **does not** confer a life-long immunity.
- Patients who recover from an attack need to have an active immunization schedule.

Gas Gangrene (Clostridial myositis)



Aetiology

Gas gangrene is an **acute** spreading **gangrene**, associated with **gas** formation and profound **toxaemia**.

- Due to infection of extensive wounds by the **anaerobic spore-bearing, bacillus Clostridium perfringens** (Welchii) & other Clostridia species.
- Gas gangrene is closely associated with grossly contaminated war injuries.

Predisposing factors

- Lacerated wounds involving **bulky muscles** as in the gluteal area and thigh.
- Presence of **foreign bodies** or devitalized tissues.
- **Ischaemia** of the **muscles**, e.g. due to injury of the main vessels, tight bandages or casts or sutures under tension.
- Infection by **aerobic bacteria** which make the medium suitable for the anaerobic Clostridia.
- Gas gangrene may follow **above knee amputation** especially in elderly persons who may suffer from **faecal incontinence** leading to infection of the stump.



Pathophysiology

Clostridia proliferate and produce toxins that **diffuse** into the surrounding tissue and destroy local **microcirculation**. **Alpha toxin**, a necrotizing **lecithinase** facilitates its diffusion, but other toxins including **collagenase**, hyaluronidase, leukocidin, protease, lipase and **haemolysin** also contribute. Clostridial toxins induce:

- Muscle **necrosis** and gas production.
- **Haemolysis** and mild jaundice.
- **Degenerative** changes in the liver, kidneys and adrenals.



Clinical Picture

Incubation period: varies from few hours to few days.

General Examination: (shock like -- toxic like)

- The patient is **pale**, anxious and apprehensive.
- The temperature may be raised, but **tachycardia** is a constant sign.
- The **hands** are cold and clammy.
- A tinge of **jaundice** may be present and there is **oliguria**.
- In severe cases the patient is **shocked**.

Local examination:

- **Pain** and **numbness** in the affected area.
- The wound is **swollen** and there may be **crepitus** with gas bubbles.
- A **sanguineous discharge** of a characteristic odour may exude from the wound.
- The affected muscles show **brick red** then **greenish** and finally **black** discolouration.
((They do not contract on pinching and do not bleed if cut.))
- The skin overlying may show greenish or black **discolouration** and there may be multiple **blebs full of a foul smelling, dark fluid**.



Differential Diagnosis

- Necrotizing fasciitis.
- Surgical emphysema, which is caused by the presence of gas under the skin. It is not associated with signs of toxæmia or peripheral circulatory failure and the wound looks healthy.



Prevention

All Clostridial infections are preventable.

- Wound debridement. Wounds occurring outdoors and contaminated with soil, foreign bodies, faeces or dirt or associated with extensive muscle injury should be examined under anaesthesia. All dead muscles (do not bleed on cutting and do not contract on pinching) should be excised and the wound is cleaned, and left open.
- Antibiotics especially **penicillin** are valuable but are in no way a substitute for adequate surgical debridement.



Treatment

A. Wound management: This is the most important step. Under **general** anaesthesia the wound is opened and all dead tissues are excised. Tight fascial compartments are decompressed. Dead muscles are adequately debrided. The deep fascia and skin are left open. When there is diffuse myositis with complete loss of blood supply or when adequate debridement leaves a useless limb, **amputation** becomes necessary.

B. Antibiotics: **Penicillin, 20-40 million units/day**, is given intravenously. In patients allergic to penicillin, clindamycin or metronidazole can be used. In mixed infections one can use multiple antibiotic combinations. Anti gas gangrene serum is no more used in modern surgical practice.

C. Hyperbaric oxygenation may be recommended.



Prognosis

- **Mortality** rate is about **20%**.
- Limb salvage (saving): With Clostridial myositis, is **not favourable**, the myonecrosis added to the original injury render it essential in many cases to perform a life-saving amputation.



Diagnosis

- Depends mainly on **clinical appearance** of the wound & the presence of large Gram-positive rods on **stained smears** of exudate or tissue.

Chronic Specific Infections Surgical aspects of tuberculosis



Introduction

Tuberculosis used to be one of the world's greatest killers. In developed countries, the recent rising incidence of "Acquired Immunodeficiency Syndrome (**AIDS**)" was accompanied by increased incidence of tuberculosis among those affected.



Aetiology

Organisms

Tuberculosis is caused by Gram-positive acid-fast rods (bacilli). There are two types of tubercle bacillus, the bovine and the human. The bovine bacillus, carried in infected milk, was a common cause of non-pulmonary tuberculosis in old days. The human bacillus is now the commonest cause of tuberculosis in man.

Mode of infection

- **Inhalation:** Direct aspiration of infected droplets from a patient suffering from open pulmonary tuberculosis is the commonest mode of infection.

- **Ingestion:** The organisms are swallowed in milk from **infected cows**.



Pathophysiology

The characteristic lesion of tuberculosis is the **tubercle**.



Microscopic

The tubercle consists of bacilli surrounded by:

- Epithelioid cells occupy the central part. They are oval.
- Lymphocytes. The lymphocytes are arranged in a circular zone near the periphery.
- Langhans giant cells are often arranged in a horse-shoe manner.

Fate of the tubercle

- **Caseation.** This is a form of coagulative necrosis affecting the epithelioid cells near the center of the tubercle.



Aspiration tuberculous pus (caseous material), although called cold abscess it is actually not cold, and not an abscess. It is slightly warm and contains caseous material.

- **Fibrosis.** When the host's resistance is high, the tubercle is surrounded by fibrous tissue and undergoes calcification.

- **Hypertrophic tuberculosis:** Here there is overgrowth of granulation tissue with scanty giant cells and no caseation. The commonest example is ileocaecal tuberculosis.



Spread

- Direct extension to adjacent structures.
- Lymphatic spread to regional lymph nodes and then to other lymph node groups or other tissues, e.g., tabes mesenterica from intestinal tuberculosis.
- Through natural passages such as in the urinary tract, air passages and bowel.
- Haematogenous spread leading to distant foci or miliary tuberculosis.



Clinical Picture

Tuberculosis usually affects **children and young adults**. It is commoner among the poorer classes.

Generally

Manifestations of tuberculous **toxaemia** may be evident and include : loss of appetite, loss of weight, night fever, night sweats and tachycardia.

Locally

Local manifestations depend on the **affected site**. The following lesions are characteristic of tuberculosis

- A Cold abscess presents itself as a **soft fluctuating mass** that lacks the signs of acute inflammation, unless there is secondary pyogenic infection.
- A tuberculous ulcer has an irregular outline, **undermined cyanotic edge** and deficient pale granulation tissue in the floor.
- A tuberculous sinus usually follows rupture of a deep abscess. It is usually unhealthy scars.



Investigations

The following is a general outline to the diagnostic tests used in tuberculosis, additional tests are used according to site.

- Bacteriological examination of exudates, discharges and body fluids for tubercle bacilli.

This includes staining techniques, e.g. Zeihl-NeeJsen and culture on LowensteinJensen medium.

- **(PCR)** is an accurate and fast test. It can detect even the smallest amount of specific DNA of the organism in tissue samples or discharge.

- **Tuberculin skin tests.** A negative result excludes tuberculosis, but a positive result is of little significance.

- **Erythrocyte sedimentation rate (ESR)** elevated but is not a specific test.

- **X-ray** examination, e.g., of the lungs, bones, and kidneys may show characteristic changes.

- **Biopsy** for histopathological examination may be needed to establish the diagnosis.



Treatment

General

• **Chemotherapy.** It is always recommended to prescribe a combination therapy of at least two drugs.

The commonly used drugs now are **isonicotinic acid hydrazide (INH)**, **rifampicin**, **pyrazinamide** and **ethambutol**. Streptomycin is less commonly used because of its toxicity. Treatment should extend for at least nine months.

• **Improving the general health.** Tonics, vitamins, good diet and open air are needed.

Local

Local management depends on the site affected, e.g. repeated aspiration for cold abscesses.

Transplantation

Organ Donation



Donor types:

[1] Living donor: Paired organs e.g. kidney & vascularized segmental pancreatic & liver transplantation.

[2] Cadaver donors: Avoids the risk of complications, which may occur to the living donors & has the advantage that multiple organs can be taken from one cadaver and transplanted to many pts with brain stem death. Criteria of brain stem death are:

1. Deep coma (no response to external stimuli).
2. Bilateral dilated fixed pupils.
3. Absence of all reflexes (gag, corneal, cough, vestibulo-ocular). Cough reflex is checked via bronchial stimulation by a catheter in the ETT, Vestibulo-ocular reflex by 50 ml ice-cold water injection into each external auditory meatus.
4. Inability to maintain the vital signs for 3 minutes without artificial means.
5. Flat EEG in all channels (i.e. absence cortical activity).

Organ Preservation:

1. Cooling (surface or perfusion) down to 0-4°C to reduce tissues metabolism.
2. Perfusion of a special fluid medium to prevent cellular edema & maintain vitality (Wiscounsinn University solution).

× Exclusions: «Should be repeated after 24 hrs & done by a separate physician not involved in the transplantation procedure»

• Sedatives, Hypnotics, Muscle relaxants.

• Hypothermia which induces deep coma.
«Core body temp. should >35°C»

• Metabolic Abnormalities (Hypoglycemia, Hyperglycemia, ↑↑ Na, ↓↓ Na, Uremia, Hepatic encephalopathy)

• Alcohol intoxication

Renal Transplantation

Hepatic Transplantation

Pancreatic Transplantation

Indications

All cases of end-stage renal disease which may be 2ry to:

1. Glomerulonephritis (most common cause).
 2. HTN, Diabetes.
 3. Chronic Pyelonephritis.
 4. Polycystic disease.
 5. Lupus nephritis.
 6. Obstructive uropathy.
 7. Congenital nephrotic \$.
- Early transplantation even before starting hemodialysis if a related living donor is available and willing.
- If the living related donor is not available, the patient is immediately placed on a cadaver donor list (not legalized in Egypt).

1. In children:
 - Cirrhosis due to Biliary atresia, Congenital hepatic fibrosis/cirrhosis.
 - Metabolic disorders as Glycogen storage disease, α -1antitrypsin deficiency, Galactosemia & Histiocytosis.
2. In adults:
 - Cirrhosis: 1ry & 2ry biliary cirrhosis, Chronic active alcoholic cirrhosis (relative).
 - Metabolic: Hemochromatosis, Wilson's disease & Budd-Chiari \$.
 - Neoplastic: The tumor should be localized and there should be no evidence of metastases.

1. Combined renal & pancreatic transplantation in Type I insulin dependent DM with renal failure.
2. Pats with previous renal transplantation suffering from severe DM endangering the grafted kidney.

Renal Transplantation	Hepatic Transplantation	Pancreatic Transplantation
Technique		
1. The grafted kidney is placed in an extra peritoneal position in the iliac fossa (preferably the right one). 2. The arterial anastomosis is performed between the renal artery and the external iliac (end to side) or internal iliac (end to end) artery. 3. The venous anastomosis is performed between the renal vein and the external iliac vein. 4. The ureter of the grafted kidney is anastomosed to the patient's urinary bladder using submucous tunnel technique as an antireflux procedure.	Orthotopic Liver Transplantation: The liver of the recipient is removed. The new cadaveric liver graft is placed in the same position of the original liver & the following anastomoses are performed in order: 1. Suprahepatic IVC. 2. Infrahepatic IVC. 3. Portal vein of recipient to donor. 4. Hepatic artery of recipient to donor. 5. Common bile duct of recipient to donor. Nowadays, segmental liver transplantation from living donors is being successfully performed. - Left lateral segment is transplanted in children, right or left lobes in adults. - Preoperative MRCP, Triphasic CT are important to study biliary tract anatomy in details for donor safety. - Recipient should have a liver graft weighing 1% of his body weight. - Graft size can be preoperatively estimated using donor CT volumetry.	There are 2 types: 1. Vascularized transplantation: Whole or segmental pancreatic transplantation is performed. i. Venous anastomosis is anchored to either portal or systemic venous circulation. ii. Exocrine secretions are drained either to jejunum «Roux-en-Y loop» or to lumen of urinary bladder. 2. Islets of Langerhans implantation (under study).
Complications		
1. Technical complications: Arterial & venous occlusion or stenosis, Bleeding & Leakage. 2. Rejection. 3. Recurrence of the original disease in the grafted organ. 4. Complications of immunosuppression.		
Results		
1. Cadaveric donor: 1-year graft survival is 95%, 5 year is 60%. 2. Living related donor: 1-year graft survival is 98%, 5 year is 90%.	1-year graft survival is 85%, 5 year is 50%.	1-year graft survival is 45%.

Heart & Lung Transplantation

Bone Marrow Transplantation

Indications

Heart:

- Ischemic cardiomyopathy beyond revascularization (45%).
- Idiopathic cardiomyopathy (55%).

Lung:

- Emphysema.
- Pulmonary fibrosis.

Both:

- Cystic fibrosis.
- 1ry Pulmonary HTN with severe LV dysfunction.

- Severe Aplastic anemia.
- Some leukemias.
- To strengthen the depleted BM weakened by potentially curative doses of radiation or chemotherapy.

Complications

- Complications of immunosuppression.
- Rejection.
- Graft atherosclerosis.

Graft vs. Host reaction: Immune response by the transplanted BM against the recipient. BMT should be confined to HLA identical siblings.

Technique

- High dose chemotherapy, Whole body irradiation immediately prior to grafting to inhibit rejection.
- BM is aspirated by multiple punctures of the iliac crest from a living donor.
- IV infusion into the recipient.

Intestinal Transplantation

In extensive intestinal resections:

- MVO.
- Crohn's disease.
- Volvulus Neonatorum, Intestinal atresia in infants.
- Radiation enteritis.

Types Of Graft Rejection

Type	Onset	Mediator	TTT	Prognosis
Hyperacute	Immediate	Humoral ABO	Graft removal	Graft loss
Accelerated	2-5 days	Humoral or Cellular	Graft removal	Graft loss
Acute	1 st month	Cellular	High dose steroids, OKT3	Good
Chronic	Several months	Cellular ± Humoral	Retransplantation (Irreversible)	Bad

Principles Of Immunosuppressive Therapy

According to their mode of action, drugs used in clinical immunosuppression are classified into 2 groups:

[A] Drugs which deplete circulating lymphocytes:

1. **Corticosteroids.** «Also inhibit IL formation»
2. **Anti-lymphocytic globulin (ALG), Antithymocytic globulin (ATG).** «Complement mediated lysis of lymphocytes»
3. **Monoclonal antibodies (OKT3):** «Specific against T-cell receptors»

[B] Drugs which inhibit lymphocyte activation or proliferation:

1. **Cyclosporine:** It suppresses T-cells & blocks release of IL-2.
Side effects: Nephrotoxicity, Hepatotoxicity, Neurotoxicity, HTN & ↑↑ K.
2. **Tacrolimus (FK506):** It arrests T-cell activation and it is 100 times more potent than cyclosporine.
Side effects: Similar to cyclosporine.
3. **Azathioprine:** It is metabolized to 6-mercaptopurine that inhibits the division and differentiation of immunocompetent lymphocytes.
Side effects: Hepatotoxicity & Leucopenia.
4. **Mycophenolate mofetil (Cellcept):** Prevents lymphocytes proliferation.
Side effects: Leucopenia & Thrombocytopenia.
5. **Rapamycin (Sirolimus):** Prevents IL-2 cellular proliferation.
Side effects: Dyslipidemia.

In addition to the previous specific side effects all immunosuppressive drugs have 2 more side effects:

1. Infections: Various viral, bacterial or fungal infections may arise in the respiratory or urinary tracts, intra-abdominally or at cannulation sites.
2. Neoplasia: Prolonged immunosuppression predisposes to the development of skin cancer, lymphomas and cervical carcinoma

Methods Of Immunosuppression

[A] Induction immunosuppression:

The first two weeks after transplantation are very important in preventing the lymphocytes from attacking the transplanted organ. This is achieved by:

1. Corticosteroids (Large Doses).
2. Azathioprine.
3. Antithymocyte globulin (ATG).

[B] Maintenance immunosuppression:

1. Cyclosporine.
2. Small doses of corticosteroids.
3. Azathioprine.



Organ Transplantation Definitions

- **Autografts:**

Same individual is donor and recipient.

- **Isografts:**

Tissue transfer from an identical twin, no rejection.

- **Allograft:**

Donor and recipient are genetically dissimilar but of the same species.

- **Xenograft:**

Donor and recipient belong to different species (animal to man) not practical.

[C] Anti-rejection treatment:

1. Selective destruction of subsets of T-lymphocytes responsible for graft destruction (OKT3 is a monoclonal antibody against T-lymphocytes carrying CD3 receptors).
2. High doses corticosteroids.

How Cancer Arises?

Gene mutations:

- Malignant transformation of a cell occurs through accumulation of mutations in specific classes of genes within it.
- Two gene classes, together constitute only a small proportion of the full genetic set, play major roles in triggering cancer, in their normal structure, they ensure normal controlled cell growth & division.

1. Proto-oncogenes:

- Encourage such growth. When mutated, proto-oncogenes can become carcinogenic oncogenes e.g. erb-B involved in development of Glioblastoma & Breast cancer.

2. Tumor suppressor genes:

- Normally inhibit abnormal cell growth and division. They contribute to cancer when they are inactivated by mutations. Examples are:
 - P53 suppressor gene is involved in a wide range of cancers.
 - BRCA1 suppressor gene is involved in breast and ovarian cancers.
 - BRCA2 suppressor gene is involved in breast cancer.

Escaping the body defense mechanisms:

- The human body contains about 30 trillion cells; thousands of them become mutated and cancerous every day.
- Our bodies are equipped with checking systems that continuously eliminate them, and only rarely does a cancer cell continue to live and to replicate producing a frank malignancy.
 1. Apoptosis (Programmed cell death) if some of the cell's essential components are damaged.
 2. Immune system recognizing & killing mutated cells from their abnormal proteins.

Etiology of cancer: Cancer seems to arise from the effects of 2 different kinds of carcinogens.

1. Agents that damage genes involved in controlling cell proliferation and migration. These initiate the malignant transformation (oncogenesis):

- a. Chemical & physical carcinogens. b. Viruses & diet. c. Idiopathic.

2. Agents that selectively enhance the growth of tumor cells or their precursors, e.g. hormones:

- a. TSH stimulates growth of papillary carcinoma of the thyroid.
b. E & P stimulate growth of breast cancers that have their receptors.
c. Androgens stimulate growth of prostate cancer.

Chemical carcinogens:

- Tobacco smoke (URT, Lung «8 times», Esophagus, Bladder «2 times», Pancreatic carcinomas) & Occupational carcinogens (Asbestos «Shipyard & Insulation workers» → Lung mesothelioma, Aromatic Amines «Petrochemical workers» → Bladder transitional carcinoma, Diesel exhaust «Garage workers» → Bronchial carcinoma, Soot «Chimney sweepers» → sq.cell carcinoma).

Physical carcinogens:

• Continuous mechanical irritation. «Gallstones → Gallbladder carcinoma»

• Ionizing radiation:

α, β, γ rays dislodge ions of water forming free radicals → DNA damage.

Bone tissue absorbs more radiation due to its density → ↑↑ Incidence of Leukemias.

Thyroid «neck irradiation in childhood», Breast, Lung cancers are also relatively common)

• UVR. «↑↑ Incidence of basal, sq. carcinoma & melanoma. Melanin in the epidermis is protective against it».

Viruses:

• Both RNA and DNA viruses but the commoner cancer-causing pathogens are the DNA viruses.

• Human papilloma viruses type 16 & 18 «STDs» can lead to cancer of the cervix, and anus.

• Hepatitis Band C viruses can cause hepatocellular carcinoma.

• EBV can cause nasopharyngeal carcinoma & Burkitt's lymphoma.

Diet:

• Animal (saturated) fat & red meat are strongly linked to malignancies of the colon and rectum.

• Alcoholics' consumption increases risk of cancer of Upper GIT & causes cirrhosis → HCC.

Idiopathic: Aging → ↑↑ Free Radicals → Gene damage. Vitamins A, C, E present in vegetables are antioxidants.

Inherited: Familial Polyposis, MEN.

Stages of cancer development:

1. Hyperplasia: the altered cell and its descendants continue to look normal but they reproduce too much 2. Metaplasia: change of 1 type of epithelium to another.

3. Dysplasia: in addition to proliferating excessively, offspring appear atypical in size, shape and orientation. Cells have:

- a. large distorted nuclei with high nuclear/cytoplasmic ratio.
- b. high mitotic activity.
- c. probably multiple nucleoli.

4. In situ cancer.

5. Invasion.

6. Metastasis.

Cancer grading:

Grading is a measure of tumor aggression. This largely depends upon its differentiation.

• Well-differentiated tumors.

• Moderately-differentiated tumors.

• Poorly-differentiated and undifferentiated tumors.



Spread of cancer:

• Properties that allow metastasis:

1. Defective cell adhesion.
2. Tumor angiogenesis.
3. Production of proteolytic enzymes that digest the basement membrane, capillary & lymphatic walls.
4. They escape apoptosis.

• Modes of metastasis

- a) Local spread
- b) Lymphatic spread. «By Embolization or Permeation»
- c) Blood-borne spread. «Lung is the commonest site then Liver»
- d) Trans-celomic spread.

Main Biological Features of Cancer cells:

Feature	Main Mechanism
Autonomous growth	Cell cycle becomes much more rapidly activated by cyclins that enhance growth & inhibit cycle checkpoints.
Ignoring antiproliferative signals	Signals of Tumor suppressor genes «p53 or TGF- β »
Invasion & Dissemination	Loss of adhesion molecules as cadherins that bind cells together. Loss of integrins that bind cells to the surrounding intercellular matrix.
Aversion to proptosis	Mutation of Bcl-2 gene family which mediates apoptosis.
Increased angiogenesis	Increased expression of VEGFs.

Diagnosis:

[A] Early detection of asymptomatic cases (screening) e.g.:

1. Annual soft tissue mammography for females at risk of breast carcinoma.
2. Annual colonoscopy for patients with ulcerative colitis at risk of colorectal cancer.
3. Annual prostate specific antigen (PSA) for men above fifty.

[B] Diagnosis of symptomatic cases:

The aim is to diagnose the disease and to determine its extent (stage).

1. Thorough clinical assessment.
2. Radiology & endoscopy.
3. Tumor sampling by Tissue biopsy & Cytology.
4. Tumor markers:
 - Many malignant tumors secrete certain oncofetal proteins e.g.:
 - a. α -feto protein: Hepatocellular carcinoma & non-seminomatous testicular tumors.
 - b. Carcinoembryonic antigen: In carcinoma of gastrointestinal tract, pancreas and breast.
 - c. Prostate specific antigen: Is raised in prostatic carcinoma.
 - d. CA 15-3: Is raised in carcinoma of the breast.
 - e. CA-125: Is raised in carcinoma of the ovaries.
 - f. CA 19-9: Is raised in carcinoma of the GIT & pancreas.
 - g. Thyroglobulin: Is raised in carcinoma of thyroid.
 - Tumors from endocrine glands, especially if benign, secrete hormones of the glands from which they arise. Examples include:
 - a. Pituitary tumors: Secrete trophic hormones as ACTH.
 - b. Pancreatic islet tumors: Secrete insulin, glucagon or somatostatin.
 - c. Parathyroid tumors: Secrete PTH.
5. Isotopic scanning is useful in detecting small metastases. «Fluoro-desoxy-glucose detects tumors with high glucose metabolism»
6. Laparoscopy: Peritoneal nodules, Small liver metastases with PET-CT scan.

TTT:

[A] Prophylactic ttt:

- Certain precancerous lesions are well known and their eradication can prevent the development of cancer.
- A well known example is familial polyposis of the colon which is 100% premalignant. Total proctocolectomy protects the patient from the subsequent development of large bowel cancer.

[B] TTT of established cases:

«By a team of surgeon, radiotherapist, medical oncologist»

1. Early (equivalent terms = loco-regional, potentially curable, operable):

- There is no evidence of distant spread & Cure is possible if this local disease is eradicated.
- Treatment is radical i.e., aims at cure & is by loco-regional modalities i.e. surgery, radiotherapy or a combination of both.
- Adjuvant treatment of systemic modalities such as chemotherapy is indicated if there is a high possibility of systemic microscopic spread in distant sites as in ↑↑ number of involved LNs.

2. Late (equivalent terms = systemic, incurable, inoperable) cancer:

- There are distant metastases & Cure is not possible.
- Treatment aims at palliation of the patient's symptoms & is done by systemic modalities as chemotherapy and hormonal treatment.
- Surgery or radiotherapy is sometimes needed to palliate local symptoms. Surgical excision is the best in GIT.
- Pain relief is accomplished by the administration of non-steroidal anti-inflammatory agents or narcotic analgesics. In severe cases neurosurgical procedures to interrupt pain pathway

[C] Individual modality treatment includes:

1. Surgery:

- **Primary tumor:** Radical surgery
- **LNs:** Block excision.
- **Precautions:** During surgery care is taken to avoid spillage of malignant cells.
- **Advantages:** Surgical excision is both quick & effective & is also the one method of therapy that offers the opportunity to confirm that a tumor has been fully excised through safety margins confirmed by a pathologist.
- **Disadvantages & limitations:** Surgery may produce functional & cosmetic disabilities & can't be applied if the tumor is fixed to a vital structure or if it has produced distant spread.

2. Radiotherapy:

- May replace surgery or may be given in addition.
- Method: Powerful X-rays, γ rays, Electrons or Heavy particles by Teletherapy or Brachytherapy.
- **Advantages:**
 - a) It can preserve the anatomical structures that surround a cancer growth.
 - b) It can destroy microscopic extensions of cancerous tissue around a tumor.
 - c) It is a safer option for older, frailer patients.
 - d) Patients treated with radiation usually do not require

THE BREAST

Congenital Anomalies

Anomalies of the nipple:

1. Athelia:

- Absence of a nipple: very rare and is usually associated with absence of the breast (Amazia).

1. Polythelia:

- Supernumerary nipples : occur anywhere along either or both the mammary ridges (mammary lines) which extend from the axillae to the groins >> may be mistaken for a mole or a wart.

Anomalies of the breast:

1. Amazia:

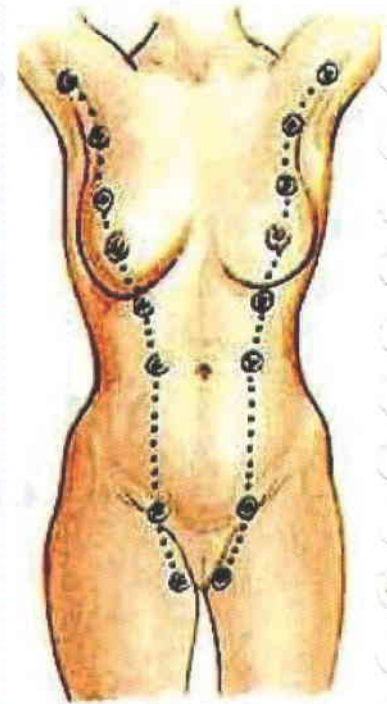
- Absence of the breast, usually unilateral and is often associated with absence of the sternal portion of the pectoralis major.

2. Polymazia:

- Supernumerary breasts due to persistence of extramammary portions of the mammary ridge.
- They may occur in axilla (accessory breast), groin, or even the thigh.
- They may function during lactation.

3. Infantile gynaecomastia:

- Diffuse enlargement of the Infant's MALE breast (unilateral or bilateral).
- Due to the effect of circulating maternal sex hormones.
- It is usually reversible within six months so requires no treatment.



Milk lines

Breast Trauma

Traumatic fat necrosis

Pathophysiology

Blunt breast trauma cause death of some fat cells>> The liberated fatty acids combine with calcium>> calcium soaps.

The result is one of two forms:

- **A CYST:** that contains thick oily fluid is formed.(more common)
- **A Hard MASS:** (less common):
 - Differentiated from carcinoma by biopsy.
 - If the lesion is excised, cut section shows a characteristic CHALKY WHITE appearance without the yellow specks and gritty texture of carcinoma.



- The breast is a modified sweat gland.
- Blunt trauma to the breast produces two lesions clinically difficult to distinguish from carcinoma:
 - haematoma, and
 - traumatic fat necrosis.
- Sometimes it is the trauma that draws the patient's attention to an already existing carcinoma of the breast.

Inflammations Of The Breast

Acute Lactational Mastitis

And Breast Abscess



Incidence

Acute bacterial mastitis is most common during lactation.



Aetiology

- **Causative organism:** coagulase positive *Staphylococcus aureus*.
- **Source:** from the mouth of the suckling infant.
- **Mode of transmission:** nipple cracks and the openings of the lactiferous ducts. Much less common is the bloodborne infection.
- **Pathogenesis:** Staph → induce clotting of milk in the ducts → obstruction and stasis (milk engorgement).



Pathophysiology

- **Site:** Infection affects any part of the breast and is at first diffuse.
- **Stages:**
The disease usually starts by milk **ENGORGEMENT**, which if not properly treated will lead to → **ACUTE MASTITIS**.
If not treated → *Staphylococci* produce necrosis and pus formation causing a multilocular **ABSCESS**.



Clinical Picture

1. Stage of milk engorgement:

- **Site:** This may affect the whole or a sector of the breast.
- **Symptoms:** a dull aching pain.

-Signs:

- 1- persistent pyrexia (General).
- 2- enlargement and induration of the breast (Local).

2. Stage of acute mastitis:

- **Symptoms:** The pain gets worse.

- Signs:

- Higher Pyrexia (General).
- Diffuse swelling, redness, induration and tenderness (Local).

3. Stage of acute abscess:

Suppuration is diagnosed if: 6(P) then (C)hronicity

1. **P**ain is throbbing.
2. **P**yrrexia is hectic.
3. **P**hysical signs get localized in the breast.
4. **P**itting oedema is elicited in the area of the skin overlying the abscess.
6. **P**ersistence of local signs of inflammation for more than 5 days or of severe systemic upset for more than 2 days **AFTER** full antibiotic treatment.

4. Stage of Chronic breast abscess.



• In 1st stage there are NO physical signs of inflammation.

• Breast abscess is one of the sites where the surgeon should not wait for fluctuation to diagnose it.



1. Before the development of an abscess:

The condition can be medically treated:

- An antibiotic against staphylococci is administered, e.g. (amoxicillin clavulanic acid).
- Support of the breast helps to lessen pain.
- Local heat (warm fomentation).
- weaning (controversial):
 - If the baby has been nursed for more than 9 months → Advise her to wean.
 - The agent in common use cabergoline (Dostinex®) 0.25 mg/12 hours for two days.
 - If the baby is younger, the patient is asked to use the healthy breast for feeding and to regularly empty the inflamed breast by squeezing and by a breast pump until resolution of infection then she can use both sides.

2. Abscess: is treated by incision and drainage.

- Under general anaesthesia.
- a circumferential incision is made over the most tender area to release the pus which is sampled for culture and sensitivity.
- The surgeon's finger breaks down the septa between the loculi to form a single cavity.
- A drain is brought out through the wound, or at the most dependent part of the breast.
- Antibiotic therapy may be continued for a few days, and the drain is removed when the drainage stops.

Non lactational mastitis

- The commonest type of non lactational mastitis is that which complicates mammary duct ectasia.
- Inflammation produces a painful, tender, and red periareolar swelling.
- Anaerobes are commonly responsible for the mastitis, and it is, therefore customary to use metronidazole or dalacin C in combination with amoxicillin clavulanic acid.
- Spontaneous or surgical drainage of such an abscess may be followed by the development of a mammillary fistula at the margin of the areola. These fistulae require surgical excision.

Chronic Inflammation Of The Breast

Mammary Duct Ectasia (Plasma Cell Mastitis)

Aetiology is not known.

Pathophysiology

The disease is characterized by:

1. Dilatation of major ducts filled with pultaceous creamy (**cheesy**) secretions.
2. Associated with periductal inflammatory reaction in which round cell (**plasma cells**) infiltration predominates.

Clinical Picture

1- Asymptomatic.

2. Nipple discharge: from one or more ducts. it may be blood stained, serous, creamy white, or yellow.

3. Retraction of the nipple: due to shortening of the ducts.

4. Acute inflammation(mentioned before).

5. Recurrent and chronic inflammation: breast abscess near areola.

Treatment

A- For non complicated cases: antibiotics (especially for anaerobes) and follow-up are recommended.

B- Complicated cases: with a mass or recurrent abscess or fistula are treated **surgically by excision** of the major ducts through a circumareolar incision. Nipple inversion is also corrected.

Because of the predominance of plasma cells, it is termed 'plasma cell mastitis'. The affected area is hard and may be associated with skin dimpling and nipple retraction simulating a carcinoma.

Chronic Pyogenic Breast Abscess

This is the result of improper treatment of an acute abscess of the breast.

ETIOLOGY:

- 1- In-adequate surgical incision.
- 2- ANTIBIOMA: If the abscess had been treated by prolonged use of antibiotics . The bacteria are killed, but the pus remains in the breast with excess fibrous tissue formation (sterile pus).

Features:

- 1- The breast abscess becomes chronically thickened with pus and fibrosis → There may be nipple retraction or skin puckering, and so the condition may simulate carcinoma. However, a chronic breast abscess is more painful and is accompanied by a low grade pyrexia.

Investigations:

Ultrasound examination and needle aspiration are helpful to differentiate both conditions.

Treatment:

A chronic breast abscess is surrounded by a thick fibrous wall and the proper treatment is **excision**, not only simple incision.

Tuberculosis Of The Breast

This is a rare disease that is always associated with active pulmonary tuberculosis, or is secondary to tuberculous cervical lymphadenitis.

Features:

- 1-The disease either presents as **multiple cold abscesses and sinuses**.
- 2-The **axillary nodes** are enlarged and matted, and manifestations of tuberculous toxæmia are present.

Investigations:

The diagnosis is based on BIOPSY → characteristic tuberculous granuloma.

Treatment :

- by antituberculous drugs.
- Mastectomy is reserved for patients with resistant infection.

Fibrocystic disease of the breast

(ANDI OR FibroAdenosis OR Cyclic Mastalgia OR Sector Mastitis)

Aetiology

- The exact cause is not known.
- Abnormal response to Estrogen cyclic changes may be a factor, evidenced by The prevalence of the disorder in women aged 30-50 years, and its rarity after the menopause.

Pathophysiology

Site: The upper outer quadrant of the breast is the commonest site.

Microscopic

A mixture of the following with a variable proportion:

1. **Adenosis:** non-neoplastic hyperplasia of glands → multiplication of acini.
2. **Epitheliosis:** is solid epithelial hyperplasia within the small ducts. When extensive it forms intraductal papillary growths and is termed papillomatosis.
3. **Papillomatosis:** extensive intraductal papillary growths.
In rare cases there is **“atypical epithelial hyperplasia”** which is accompanied by a higher possibility of developing breast cancer.
4. **Fibrosis:** is the replacement of elastic and fatty tissue by fibrous tissue. When fibrosis is extensive (**sclerosing Adenosis**) it may simulate carcinoma on mammography and needs Tru-cut needle biopsy for diagnosis.
5. **Cyst formation:** due to obstruction of ducts by epitheliosis. They may be small (microcysts), or large (macrocyts). They are lined with epithelium and are filled with clear yellow, or sometimes brownish fluid.

• This is the most frequent disorder of the breast.
The acronym ANDI stands for “Aberrations of Normal Development and Involution”.

• It is not considered a disease, but a variation or aberration of the normal changes that take place in a healthy breast with the menstrual cycles, pregnancy, lactation, and menopausal involution.

• A cyst may enlarge and hemorrhage occur inside → Occult blood → Called Cyst of “Bloodgood”.



"A palpable thickening may disappear when the patient is re-examined one week after the menstrual cycle. The differentiation from carcinoma is discussed under the differential diagnosis of breast cancer".

Criteria of Benign cysts

1. If the fluid is not blood-stained.
2. The mass disappears completely after aspiration (no residual mass).
3. Does not recur within two weeks.
4. Absence of any malignant cells on Cytology.



Clinical Picture

1. **Cyclic painful nodularity** (small lumps) is The **most frequent** complaint, it may be unilateral or bilateral. The lady usually observes this in relation to her cycles.
2. **Cyclic Mastalgia** (breast pain) is a common presentation. This is usually exacerbation of the premenstrual tension.
 - The typical pain occurs few days **premenstrually** and is accompanied by enlargement and increased nodularity of the breasts.
3. Nipple discharge :usually clear or yellow, but sometimes brown or green.
4. **Asymptomatic** in some ladies.
5. **accidentally felt lump:** This may be caused by a prominent cyst, aggregation of small cysts, or by the sclerosing adenosis.



Investigations

Investigations are **usually not required**, but the following are indicated to **rule out** breast cancer in suspected cases.

1. **Ultrasound and mammography:** show the cysts.
2. **Aspiration:** (to exclude malignancy)
 - a. A solid mass is aspirated for cytology.
 - b. Open biopsy is indicated when experience in interpreting cytology is not available, or if its result is not conclusive. Excised specimens are shown in.



Treatment

Treatment is individualized. **Exclusion of malignancy** and **reassurance** of the patient are most important.

- **Accidentally discovered cases:** No treatment.
- **Cysts:** are treated by aspiration. A recurring cyst is excised for biopsy.
- **Mastalgia:**
 - A- Psychological Support:** reassurance.
 - B- Breast Support:** wearing (night and day) a firm bra that gives good support and protection are usually enough.
 - C- Lifestyle support:** Giving up caffeine consumption (coffee, tea, and chocolate).
 - D- Primrose oil:** one capsule every night (relieves pain):
 - o **Topical nonsteroidal anti-inflammatory drugs:** (NSAIDs, e.g., diclofenac gel) had been proven to be effective with minimal side effects.
 - o **Prolactin inhibitor:** As cabergoline (Oostinex®) 0.5 mg/week for six weeks.

Cysts of the breast

Cysts of the breast usually arise from the duct system (acinar cysts), and occasionally they occur in the stroma (interacinar cysts).

Acinar cysts

1. Retention cysts:

- Due to blockage of the secreting mechanism, either by **fibrosis** from without or by **obstruction** from within the lumen.
- They account for more than 75% of cysts of the breast and are mostly fibroadenosis. The cyst is often solitary, and varies from one quarter to two inches in diameter.
- The fluid content may be clear and serous, thick and greenish, or creamy in colour and consistency.
- Large cysts distended with clear fluid may have blue green colour (blue-domed cysts of Bloodgood).

2. Galactocoele (milk cyst):

- Develops during or shortly after lactation.
- Probably from obstruction to one of the principal ducts.
- At first, the content is **thin and milky** and the cyst is tense and thin-walled, but gradually the milk becomes inspissated and the wall thickened and fibrous.
- The cyst forms a solitary painless swelling situated **close to the nipple** and dating from lactation. Sometimes a little milky fluid may be expressed from the nipple by pressure on the cyst.
- Excision is usually needed.

3. intracystic papilliferous carcinoma.

Interacinar cysts

Cysts arising in the stroma are rare and include:

1. Dermoid cysts.

2. Lymph cysts.

3. Blood cysts: These arise either as traumatic haematomas or as other types of cyst with bleeding into them.

4. Sebaceous cysts: These may arise in the skin near the nipple in relation to one of the **tubercles of Montgomery**.



Treatment

is usually by excision

Nipple Discharge



Aetiology

• Physiological:

- o Milk production with **lactation**.
- o Serous discharge during **pregnancy**.

• Pathological: The commonest cause is mammary **duct ectasia**.

Table Of Causes Of Nipple Discharge

Duct ectasia	Serous, creamy white, yellow, or blood stained. It may be also brown, green or black - usually multiple ducts
Fibrocystic disease	Clear, yellow, brown or green - multiple ducts
Duct papilloma	Blood or blood stained discharge from one duct
Duct carcinoma	Rare cause of blood or blood stained discharge from one duct
Contraceptive pills	Serous or milky discharge from multiple ducts
Hyperprolactinaemia	Milky discharge (galactorrhoea) from multiple ducts



Diagnosis

History and examination should provide the following information

1. Nature of discharge.
2. Association with a mass.
3. Unilateral or bilateral.
4. Single duct or multiple duct discharge.
5. The use of contraceptive pills.



Investigations

1. Test for occult blood in the discharge if it is not apparent.
2. Cytology of the discharge for exfoliated cancer cells.
3. Soft tissue mammography and ultrasonography.
4. Ductography may be useful in cases of single duct discharge. The test is not so practical and has been superseded by the simpler mammography.
5. Serum prolactin estimation in suspected cases of galactorrhoea.



Treatment

1. **A palpable mass:** should be excised for histology and treated accordingly.
2. **A single duct:** bloody discharge calls for excision of this duct by the operation of microdochectomy.
3. **Multiple ducts discharge:** with no palpable nor mammographic mass is initially treated conservatively by observation.

Breast neoplasms classification and benign tumours

Classification Of Breast Neoplasm			
Benign		Malignant	
Epithelial	Duct papilloma	Epithelial	Carcinoma
Mixed mesenchymal and epithelial	Fibroadenoma	Mesenchymal	- Lymphoma (rare) - Fibrosarcoma (rare) - Cystosarcoma - Mixed mesenchymal Fibroadenoma and epithelial

Duct papilloma



Pathophysiology

Site: Mesenchymal, a duct papilloma is usually situated in one of the main ducts near the nipple in a young woman.

Pathogenesis:

Before it becomes big enough to form a palpable lump, it may ulcerate, bleed and cause a blood stained discharge from the nipple. If it blocks the duct it may cause a retention cyst.



Clinical Picture

- 1. Bloody, or blood stained nipple discharge:** The commonest symptom.
- 2. Swelling :** accumulation of blood in the duct. It may be felt deep, or just lateral, to the areola:
 - The mass is small & fusiform, with its long axis pointing to the nipple.
 - Pressure on the swelling produces the discharge .



Investigations

- 1. Ultrasound and mammography:** to reveal the mass information.
- Ductography and ductoscopy are usually not needed.



Treatment

Treatment is by excision of the affected duct (**micro-dochectomy**) through a circumareolar incision.

- **If there is a lump:** it easily leads to the duct.
- **If there is no lump:** the duct is identified at operation by passing a blunt tipped needle through the discharging nipple opening.
- The excised specimen should be **histologically** examined.



If there is no palpable swelling, pressure on a certain point behind the areola will reveal the discharge coming out from one duct opening (Zonal pressure test).

Fibroadenoma

Fibroadenoma is the **commonest breast mass of young women**.

The usual age is between 15 and 30 years. It is a benign neoplasm.



Pathophysiology



Microscopic

- Site:

It affects both the fibrous and the glandular tissues, but the fibromatous element predominates.

- Histologically there are two types:

- **Pericanalicular (hard) fibroadenomatata:** are the usual form. These tumours are formed mainly of fibrous tissue that surrounds a few small tubular glands. They tend to be small and firm.
- **Intracanalicular (soft) fibroadenomatata:** contain more glands. They are usually larger and softer than the pericanalicular type.



Gross

the tumour may be solitary or multiple, **firm**, with **smooth** surface that may be **lobulated** in big lesions. It is **well circumscribed** and is **never attached** to surrounding tissue (freely mobile).

The cut surface reveals lobules of **whorled** white fibrous tissue which bulges out of its capsule.



Clinical Picture



Symptoms

A painless lump (or lumps): that is accidentally discovered.



Signs

- A fibroadenoma is usually **small** (few centimeters), **nontender**, spherical, **firm**, **well circumscribed**, and with smooth surface that may be lobulated.

The characteristic feature, however, is its high mobility within the breast that gained it the name **breast mouse**.



Investigations

- **Ultrasonography and digital mammography** reveal a well circumscribed lesion.
- When in doubt, **Tru cut needle biopsy** rules out malignancy.



Treatment

- **Lesions ≥ 2.5 cm:** excision and histological confirmation of the diagnosis.
- **For smaller lesions:** follow-up may be performed.

Phyllodes Tumours



Incidence

It occurs after the age of thirty years.



Pathophysiology

It is classified to benign, borderline or malignant according to cellular atypia, mitosis and stroma overgrowth.



Gross

the tumour may **stretch the skin** which may **ulcerate** and shows **dilated veins**.



Microscopic

The tumour shows compressed branching ducts like a leaf or a fern.



Treatment

Excision with 1 cm safety margin.

- If the tumour is involving the whole breast, **mastectomy** may be performed.



Phyllodes Tumours

- Previously known as Cystosarcoma phylloides.
- It is neither a cyst nor sarcoma.
- It is a rapidly growing tumour resembling intra-canalicular fibroadenoma.

Carcinoma of the Breast



Incidence

- Breast cancer is the most frequently diagnosed cancer in women worldwide (23% of total cancers).
- National Cancer Institute in Cairo recorded breast cancer to represent about 35% of all female cancers.
- However, the annual mortality rate decreased over the last decade by 1.9%, mostly due to early detection programs and advances in the adjuvant therapy.



Aetiology

The exact etiology is not known. However, the risk factors are :

1. Genetic factors: It has been proved that in **5-10%** of all cases of breast cancer, there is an autosomal inheritance of a mutant gene.

Two genes **BRCA1** (on chromosome **17**) and **BRCA2** (on chromosome **13**) are involved. These cancers usually occur at a **younger age** and are multifocal and **bilateral**.

2. Family history: The risk of developing breast cancer is increased by 1.5 to 3 fold if a woman has a **first degree** relative with breast cancer. The **highest risk** is associated with a **young first-degree relative** with bilateral breast cancer.

3. Personal history of breast cancer increases the incidence of **contralateral** breast-cancer.

4. Proliferative breast disease:

Mild or florid hyperplasia → increases the risk by **1.5-2** times

Atypical hyperplasia → increases the risk by **4-5** times

Lobular or ductal carcinoma in situ → increase the risk by **8-10** times



Most cases develop **after the age of 50 years** with a peak at 70 years.
%10-5 of cases develop under 40 years.

5. Endocrinal factors

- The total duration of exposure to endogenous **estrogens** is an important factor of breast cancer risk.
- **Early menarche** below the age of 13 years, onset of first pregnancy after the age of 30 years or **late menopause** after 55 years increases the risk of development of breast cancer.
- **Hormone** replacement therapy **slightly** increase the risk.
- **Obesity** and high intake of **saturated fatty acids** are associated with an increased risk. Steroid hormones are converted to oestradiol in fatty tissues.



Pathophysiology



Site

Most breast cancers develop from the **terminal duct lobular unit**.



Pathological Types

(A) Carcinoma in situ:

1. Ductal carcinoma in situ:

- 20-30% of newly diagnosed cancers by mammography are in situ.
- This usually presents as a density or **micro-calcification** on mammography. if not excised, will turn to invasive breast cancer.
- Treatment is by breast conservative (lumpectomy) followed by radiotherapy.

2. Lobular carcinoma in situ: (usually bilateral multicentric)

- This is usually detected after a pathological study of an excised breast lump. It is a marker that the patient has an **increased risk** to develop breast cancer in the other breast. The patient needs a meticulous follow-up.

(B) Invasive breast cancer:

1. Invasive duct carcinoma (not otherwise specified) 75% :

This was previously known as **schirrous carcinoma**.

The tumour is **hard**. The cut surface is **concave, rough, gritty, and pale grey** with prominent yellow and white flecks.

Rapid lymphatic spread occurs through the breast and there may be multiple satellite masses within the breast.

2. Invasive lobular carcinoma (5-10%):

it may be bilateral and it shows **multicentricity in 20%** of cases.

3. Medullary carcinoma (5-7%).

It has a well circumscribed border, and there is **intense infiltration by lymphocytes** and **plasma cells**. Prognosis is **favourable**.

4. Mucinous carcinoma (3%).

There is abundant accumulation of extracellular mucin.

5. Tubular carcinoma (2%).

There is a tendency to **tubular formation**. Prognosis is **good**.

6. Inflammatory carcinoma.

This is a rare **highly malignant** tumour which grows rapidly and invades the dermal plexus of lymphatics.

7. Paget's disease of the breast:

It is essentially an **intraduct carcinoma** which begins in the epithelium of a **main collecting lactiferous duct** and spreads within the epithelium **up to the skin of the nipple** and down into the breast substance.



Microscopic

There are **three** characteristic features which are:

- 1- **Paget's** cells.
- 2- **Hyperplasia** of all layers of the epidermis.
- 3- **Round-cell** infiltration of the dermis.



Spread

1. Local spread: can occur through the breast substance, overlying skin, underlying pectoralis major and serratus anterior muscles, and the chest wall.

2. Lymphatic spread: (by **embolism** and **permeation**) is mostly to the **axillary** nodes, next common is the **internal mammary** chain. Involvement of the supraclavicular nodes is considered an advanced disease.

NBs:

- **By the time** the lymph nodes are histologically affected by malignancy, one should assume that distant organ micrometastases are likely to be present, and the patient is treated accordingly.

- Lymph node affection is, therefore, **THE MOST IMPORTANT** prognostic factor.

- Obstruction of the dermal lymphatics by malignant cells produces breast skin oedema that is marked in the dependent part, i.e., the lower half of the breast. The oedematous skin is pitted at the sites of **hair follicles, sebaceous glands, and sweat glands** giving the appearance of an orange peel, hence the French term **«peau d'orange»**.

3. Blood spread:

produces metastases in the lungs, bones, brain and the liver. The **bones** commonly involved by metastases include the lumbar vertebrae, ribs, femur and the skull. Especially **the Spine**, explained by the free communication of the posterior intercostal veins with the **paravertebral plexus of veins**.



• Paget's cells are clear vacuolated cells with small dark-staining nuclei which occur alone or in clusters in the deeper layers of the epidermis.

• Previously it was thought that breast cancer spreads locally at first, then by lymphatics, and lastly by the blood stream. This view is no longer accepted, and it is now well realized that carcinoma of the breast may spread by the blood stream very early, producing micrometastases in distant organs.

Hormone Receptors

- About **60%** of breast cancers have receptors for oestrogen and are termed **“ERpositive”**.

These tumours get more active under the influence of this hormone. The tumour can be induced to regress if the patient is deprived of oestrogen, or the hormone receptor is blocked by an anti-oestrogen.

- Tumours may also have progesterone receptors.
- “” Hormonal receptor +ve tumors >>> better prognosis “”

- About **20%** of breast cancers exhibit **HER2/neu** receptors which are members of epidermal growth factor receptors involved in cell growth regulation “BAD PROGNOSIS”.

- About **30%** of patients do not have ER, PR or HER2/neu receptors (**Tripple negative** breast cancer). “BAD PROGNOSIS”.

- Overexpression of Her2/neu or triple negative type indicates a worse prognosis.



Clinical Picture



Symptoms

1. **A painless lump** : accidentally noticed by The patient while washing or looking into a mirror. An accidental trauma to the breast may attract the attention of the patient to the presence of the lump.

Much less frequently:

2. **Mild breast pricking pain , recent nipple retraction** or blood stained nipple **discharge**..

3. **Occasionally** breast cancer announces its presence by the appearance of **metastases**. The latter include an **axillary lump, backache** caused by spine metastasis, **pathological fracture, dyspnoea and pleuritic pain** caused by pulmonary metastasis, **jaundice** caused by liver secondaries, or mental changes and fits due to cerebral deposits.

4. During a **routine screening mammography** that is done for the high risk asymptomatic women.



Signs

General: Cachexia , or of distant metastases as bony, pulmonary, cerebral or hepatic metastases.

Local:

1. Breasts:

Show **asymmetry, enlargement, skin dimpling**, or **skin puckering**.

Dimpling and puckering of the skin are evident when the patient is sitting and **elevates her arms, Peau d'orange, skin nodules and ulceration** indicate a locally advanced disease.

2. Mass:

o Hard, irregular and ill-defined.

o **Restricted mobility** within the breast substance.

o Fixation to the skin, underlying muscles, or chest wall, if present, is diagnostic of carcinoma.

3. Nipple:

o Recent retraction.

o Change of direction.

4. Axillary and supraclavicular nodes: Number and mobility of palpable nodes are assessed.

• Skin tethering is caused by invasion and shortening of a ligament of Cooper, where the mass can be moved within a certain range without dimpling the skin, but outside this range the skin is dimpled.

Skin puckering, on the other hand, is caused by direct infiltration of the skin with the tumour, and the mass cannot be moved at all without moving the skin.

• It should be noticed that clinical evaluation of the axilla may be inaccurate. Impalpable axillary nodes may turn out to have tumour deposits when examined histologically after the operation.

Special clinical forms

Paget's disease of the breast:

The first symptom is often an abnormal pricking sensation of the nipple, with superficial erosion. A tumour mass may not be palpable. The lesion is commonly mistaken for eczema. The diagnosis is established by biopsy of the erosion.

Clinical differences between Paget's disease of nipple and eczema	
Paget's disease	Eczema
Unilateral	commonly bilateral
Usually occurs at menopause	Commonly occurs at lactation
No itching	Itching
No vesicles-not oozing (dry)	Vesicles-oozing
Nipple is eroded	Intact nipple
Well defined	Ill defined
A breast lump may be felt	No lump
No response to eczema treatment	Responds to treatment
Starts in the nipple	Starts in the areola

Inflammatory carcinoma:

- This is a rare aggressive form of breast cancer, which may occurs during pregnancy or lactation. There is a rapidly growing, sometimes painful, breast swelling. The overlying skin becomes red, oedematous, and warm.
- Often there is no distinct mass, since the tumour infiltrates the breast diffusely.
- The picture clinically resembles acute mastitis.
- The prognosis is poor as it is usually advanced at the time of diagnosis.

Differences between Inflammatory Carcinoma & Acute Bacterial Mastitis	
Inflammatory Carcinoma	Acute Bacterial Mastitis
Gradual onset with no or low grade fever	Acute onset with high fever
Progress is slower	Rapid progress
Involves more than one third of the breast	One breast sector is affected
Skin is dusky red	Skin is rosy or fiery red
Mildly tender or non-tender lesion	Markedly tender lesion
Non-tender axillary nodes	Tender axillary nodes
No response to antibiotics in one week is an indication for biopsy	The lesion is either cured by antibiotics or forms an abscess

Carcinoma in situ:

- Is now more frequently recognized due to the use of mammographic screening. 20% of cancers detected by screening are in situ Cancers.

Differences between Duct Carcinoma In Situ (DCIS) & Lobular Carcinoma In Situ (LCIS)		
	Duct Carcinoma In Situ (DCIS)	Lobular Carcinoma In Situ (LCIS)
Frequency	More common	Less common
Microcalcification	Present	Absent
Bilaterality and multicentricity	Rare	Common
Early detection	Possible	Less likely
Potential for invasive cancer	%50-30	It is a marker of increased risk of malignancy in the same of other breast
Treatment	As invasive cancer, but no axillary dissection	Strict follow up



Differential Diagnosis

- Cases presenting by a lump (mass): The clinical features of common breast lumps constitute 95% of breast lumps.

Clinical features of common breast lumps				
	Carcinoma	Solitary Cyst	Fibrocystic disease	Fibroadenoma
Age	Usually > 35y	35-55y	35-55y	15-30y
Pain	Painless	Occasionally painful	Occasionally painful	Painless
Surface	Irregular	Smooth surface	Indistinct surface	Smooth, lobulated if large
Consistency	Usually hard	Fluctuation is difficult to elicit, so it feels soft to hard	Firm, ill defined areas of thickening	Firm Highly mobile
Lymph nodes	Probably axillary node enlargement	Free axilla	Free axilla	Free axilla



Staging

Initial treatment is based on the clinical staging then further management is based on the pathological staging which may differ from the clinical one, e.g. lymph nodes which were clinically impalpable may be found to be histologically involved by the disease.

The two common systems of staging are:

A. International TNM staging

T = Tumour	N = Nodes	M = Metastases
T _{is} = Carcinoma in situ. Paget's disease with no palpable tumour.	N ₀ = No palpable axillary nodes.	M ₀ = No evidence of distant metastases.
T ₀ = No evidence of primary tumour.	N ₁ = Mobile palpable homolateral axillary nodes.	M ₁ = Distant metastases.
T ₁ = 2cm diameter or less.	N ₂ = Fixed homolateral axillary nodes. N ₃ Palpable homolateral supraclavicular nodes.	
T ₂ = 2-5 cm diameter.		
T ₃ = Tumour larger than 5cm.		
T ₄ = Any size with direct extension to chest wall or to skin; or inflammatory carcinoma		

B. Staging of the UICC (union international centre cancer) is now internationally approved.

It is based on the TNM staging

UICC staging			
Stage	Description	Category	5 yr survival
I	T ₁ N ₀ M ₀	Early breast cancer	93 %
II	IIA T ₂ N ₀ M ₀	Early breast cancer	72 %
	IIB T ₃ N ₀ M ₀	Locally advanced breast cancer	41 %
III	IIIA T ₁₋₃ N ₁₋₃ M ₀		
	IIIB T ₄ N _{any} M ₀		
IV	T _{any} N _{any} M ₁	Metastatic	18 %

Investigations

Aim:

- Diagnosis of carcinoma by imaging (mammography, ultrasonography) & biopsy.
- Metastatic work-up.

Breast imaging:

1. Digital mammography:

- In expert hands it is **95%** accurate in diagnosing breast cancer.
- **If combined** with Tru-cut biopsy, their accuracy of diagnosis is improved.
- useful for detecting **multifocal lesions** in the same or other side, before deciding radical surgery.

- A cancer appears as:

- A **dense opacity** that has an **indefinite outline** from which irregular spicules penetrate into the surrounding breast.
- Clustered **microcalcifications**.

2. Ultrasonography:

- It can differentiate cystic from solid lesions.
- A spiculated hypoechoic mass more deep than wide suggests a malignant lesion.
- Adding colour coded Duplex ultrasound identifies the vascularity of the lesion.

3. Magnetic resonance imaging (MRI):

- is indicated with contrast in:
- Postoperative **scarring** to differentiate fibrosis from local recurrence of malignancy.
 - Before and after **neoadjuvant** therapy to monitor response.
 - In the presence of breast **implants**.

Biopsy:

A pathological evidence of malignancy is the **corner stone** of confirming the diagnosis.

The different types of biopsy are:

1. Tru-cut biopsy

- It is done under **local anaesthesia** with special needle that cuts a core of tissue out of the tumour.
- The obtained specimen allows for **histological examination** and for assessment of the **receptors**. It is better to be performed under ultrasound guidance.



- Mammography is of less diagnostic value in young women, because the density of the lesion differs little from that of the surrounding tissue.

- Contrast enhanced digital mammography and tomosynthesis (high resolution mammography) help in these cases.

- In duplex study, Malignant lesions receive blood flow from all around with turbulent speed, whereas benign lesions receive blood flow from one side at a low speed.

- It is particularly useful in young women when mammography may not be very helpful.

- U/S guided Tru-cut needle biopsy is becoming the standard method for diagnosis of breast cancer.

2. Fine needle aspiration cytology (FNAC).

- Depends on examination of **cells** to detect criteria of malignancy in them.
- The aspiration can be done in the outpatient clinic using a **10-20 ml** syringe and a **21-gauge** needle.
- False negative results may occur in up to **10%** of cases.

3. Excision biopsy.

- The **whole mass** is excised through a circumareolar or a transverse **incision**.

The results need around **two day**.

- the **most reliable** and provides a big enough specimen to allow for hormone receptor estimation as well. It is, however, gradually **loosing popularity** in favour of the simpler fine needle aspiration cytology and Tru-cut biopsy.

4. Frozen section biopsy.

- The tumour is either **excised** or, if large, a small piece is obtained by incising it (incision biopsy).
- A diagnosis is obtained within 20 minutes.
- If the result of examination is negative for malignancy, the patient is awakened. If it is positive, the surgeon proceeds with radical surgery.

5. Stereotactic core biopsy (mammography guided)

is used for **nonpalpable** mammographically detected lesions, such as microcalcifications that cannot be seen with ultrasonography.

6. Biopsy from impalpable breast masses.

The radiologist can place a **hookwire** inside the lesion under **mammographic guidance** or ultrasound guidance. At operation, the mass is removed with the wire for histological assessment.

Metastatic work-up

To decide whether the case is M_0 or M_1

- **In early breast cancer:** chest x-ray and U/S of the abdomen are enough.
- **In locally advanced breast cancer:** more investigations may be required e.g. CT scan for the chest and abdomen, bone scan or PET scan.

Triple Assessment

This means comparing the results of:

1. Clinical examination.
2. Soft tissue mammography or ultrasonography.
3. Tru-cut needle biopsy:

If there three parameters are concordant, the surgeon can rely on the diagnosis.

If the three parameters are not concordant, further investigation is needed.

1. Early detection

Detection of breast cancer very early in the asymptomatic females.

There are two main methods for early detection of breast cancer.

2. Breast self examination (BSE)

All women over age 20 should be advised to examine their breasts monthly, **one week after the menstrual period**.

If the woman suspects the presence of a lump, skin dimpling, or recent nipple retraction, she should report to the surgeon.

3. Screening programs

High risk women are subjected to regular clinical **examination** and **mammography**. Screening mammograms are performed in the asymptomatic patient.

- consist of two standard views, **mediolateral oblique** (MLO) and **craniocaudal** (CC).

- In patients with a **family history** of breast cancer, **annual** mammograms and **semiannual** physical examinations should begin **10 years** earlier than the age at diagnosis of the youngest affected relative and no later than the age of 40 years.

- 30% of detected lesions are **ductal carcinoma in situ**.



Treatment

- Treatment depends on the **stage** of disease. There are three categories of patients:

-Early breast cancer. -locally advanced disease. -Advanced disease.

Treatment of early breast cancer:

- **Definition:** Those cases categorized as stages **I** and **IIA** in UICC staging.

- **Aim: Local eradication** of the disease . This is usually achieved by surgery in combination of some form of adjuvant therapy as outlined below.

- ONE of the following two strategies may be adopted with full explanation to the patient:

1. Conservative breast therapy:

A- Management of the breast:

- **Wide local excision** of the tumour with a **1-cm** safety margin.

- If the lesion is **close to the skin**, part of it may be excised to ensure the required safety margin.

- Indications: (S.S)

1. **Small** tumours **4 cm** and less.

2. Sometimes large lesions (up to **5 cm**) in **large** breasts.

3. The lesion should be **solitary** or multifocal (lesions present in **one quadrant**)

- Contraindications: (PCs)

1. **Pregnancy** as radiotherapy is contraindicated.

2. Extensive **microCalcification** (multi-compartmental)

3. **MultiCentric** (masses in more than one quadrant) as detected by soft tissue mammography or MRI.

4. **Collagen** vascular disease (poor tolerance to radiotherapy).

5. **Central** or periareolar tumors. No benefit from conservative surgery because nipple and areola will be removed.

B-Management of the axilla:

1. Sentinel lymph node study:

- It is indicated in **stage N₀** (clinically & radiological negative axillary LN).

- Injection of **methylene blue** 1 % or a radioactive sulphur colloid near the tumour or retroareolar will allow, identification, excision and immediate pathological examination of the sentinel lymph node.



- Conservative therapy is gaining popularity in recent years.

- It provides good results that are equal to radical mastectomy, while the breast is preserved minimizing psychological trauma.

-The term therapy is used because it includes a combination of surgery and radiotherapy.

If the node is positive for metastases → axillary clearance

If the node is negative for metastases → no further excision of lymph nodes.

N.B: The value of sentinel lymph nodes biopsy is to avoid axillary dissection in node negative patients, thus **reducing the incidence of lymphedema**.

- For N1 patient axillary lymph node dissection (**ALND**) is performed **from the start**.

- Removal of level I and level II nodes and, if grossly involved, level III nodes.

- An ALND should remove 10 or more nodes. The number of nodes identified is often pathologist-dependent.

C- Postoperative radiotherapy:

Postoperative radiotherapy is a must after breast conservative therapy.

- It is done for **5 weeks** directed to the **whole breast** with a boost at the site of the operation in order to kill any **residual malignant** cells.

D- Further management:

- **Hormonal therapy** (HT) for all hormone receptor positive cases.

- It reduces ipsilateral and contralateral breast recurrence by **40%**.

- **Tamoxifen** blocks estrogen receptors.

- **Anastrozole** is an aromatase inhibitor. It is suitable only for postmenopausal patients.

E- Chemotherapy:

- Is indicated for

- i. **Any** Positive axillary **nodes**.

- ii. Tumours more than **5 cm**.

- iii. Hormone receptor negative and **HER2/neu positive** tumours (denote aggressive tumours). The drug regimen commonly includes **anthracyclines** and **taxanes**.

Targeted therapy for HER2/neu positive cases. It is a monoclonal antibodies are given against HER2/neu receptors (**herceptin**) improves survival in this group of patients.

2. Modified radical mastectomy, MRM (of Patey):

- Indicated for:

Large, central, multicentric tumours or **local recurrence** after conservative surgery.

A- Mangement of the breast:

A skin ellipse over the tumour with at least **5 cm** safety margin, including the areola and nipple is made.

The **whole breast** including the tumour is excised.

B- Management of Axilla:

- The axilla is cleared, i.e. all lymph nodes medial to the axillary vein and the axillary fat are removed in continuity with the excised breast.

- Usually level **I and II only** are excised.

- BUT The **axillary vessels, the nerve to serratus anterior, and the nerve to latissimus dorsi are spared.**

C- Postoperative radiotherapy: is advised for:

- patients with **positive axillary lymph nodes** (Patients with 4 or more positive lymph nodes and selective patients with 1 to 3 positive nodes)
- for **T₃** patients.
- for tumours in the **medial half** of the breast.

D- Adjuvant systemic therapy: Is given in appropriate patients after completion of surgery, for **all node-positive patients** and **T₃** patients.

Treatment of intermediate disease (locally advanced breast cancer)

Giving the adjuvant chemotherapy treatment before surgery (**neoadjuvant therapy**) allows reduction in the tumour size and allows breast conservative therapy instead of mastectomy in some cases.

The same drugs used in the adjuvant protocol are used in neoadjuvant treatment. It should be noted that patients who receive neoadjuvant treatment will start radiotherapy after surgery and will not take postoperative chemotherapy any more.

Treatment of advanced disease:

This category comprises stages **III and VI** UICC, **T₄** patients, **fungation, ulceration, inflammatory carcinoma** or **recurrent** cases as well as distant metastases are included in this category.

- **Aim:** palliation and improving the quality of life.
- **Modalities of treatment:**

1. Radiotherapy

- a. It is indicated **locolly** for palliation of **pain** or **ulceration**.
- b. Systemically for **brain** and **spinal** metastases and for **bone** secondaries.
- c. **Superior vena cava** obstruction.

2. Hormone therapy

The following issues are relevant

- a. Only for hormone **receptor** positive patients, with a 60-70% response.
- b. It is more effective in **postmenopausal** women.
- c. It is **not very effective** in hormone negative patients (response, 10%), with visceral metastases or young patients below 35 years.
- d. **Tamoxifen**, the primary antioestrogen is given for **not more than five years** to avoid the risk of endometrial cancer or thrombogenicity. If no response to tamoxifen, aromatase inhibitors may be used.

3. Chemotherapy

The following issues are relevant

- a. It is the **basic treatment** of metastatic disease; response 60-80%.
- b. It can be used as a targeted therapy for **Her2/neu positive** cases.
- c. It is given for **visceral** metastases as the liver, lungs or brain, for **hormone negative** patients and failure of hormone therapy.

4. Surgery

is indicated for the following cases

- a. **Simple mastectomy** to remove an unpleasant, malodorous fungating tumour.
- b. **Internal fixation** of pathological fractures.
- c. **Urgent decompression** and stabilization of the spine if a vertebral collapse causes spinal cord compression.

Reconstructive procedures after mastectomy

There is now great tendency to offer the patient who had mastectomy, some form of reconstructive surgery so that there will be minimal cosmetic deformity. Two procedures are available.

1. **A synthetic implant:** may be inserted behind the pectoralis major muscle.
2. **A myocutaneous flap:** is used to reconstruct the breast. The transverse rectus abdominis myocutaneous flap (TRAM) is very efficient. The idea is take a transverse flap of the skin of the lower abdominal wall together with the rectus muscle with the blood supply based on the superior epigastric vessels. The flap is transposed upwards to form a breast substitute.

Management of specific problems

1. **Hypercalcaemia** occurs in patients with widespread bone metastases. Patients complain of thirst, drowsiness, and constipation. The patient is dehydrated. Serum calcium is high.
Treatment: correction of dehydration by IV fluids, together with furosemide, prednisolone and biphosphonates.
2. **Pathological fractures** Internal fixation and radiotherapy.
3. **Cerebral metastases** are treated by a combination of corticosteroids and radiotherapy.
4. **Spinal cord compression** requires urgent surgical cord decompression, with stabilization, followed by radiotherapy.
5. **Pleural effusion** commonly responds to systemic therapy and chest tube drainage. If not, local instillation of the cytotoxic bleomycin through the tube may be required.
6. **Liver metastases** are indication for chemotherapy.



Prognosis

It depends on the following factors:

1. **Involved lymph nodes:** Number, size, mobility, and location . Assessment of lymph node involvement depends on the histological examination not only the clinical evaluation which is very inaccurate.
2. **Type of the tumour:** The best prognosis is provided by the in situ carcinoma, and Paget's disease, while the worst is the inflammatory carcinoma.
3. **The T stage:** of the primary tumour. The higher the T stage, the worse is the prognosis.
4. **The presence of distant metastases:** markedly worsens the prognosis.
5. **Hormone receptor status:** Receptor positive tumours respond more often to hormonal therapy and have a better prognosis than those that are receptor negative.
6. **The site of the tumour:** **Medial half** tumour have a **worse** prognosis than those of lateral half due to early involvement of the internal mammary lymph nodes.

DISEASES OF THE MALE BREAST

Gynaecomastia

It is generalized enlargement of the male breast (unilateral or bilateral).



Aetiology

Most cases are related to an **imbalance** between oestrogens and androgens.

Physiological gynaecomastia

- Infantile gynaecomastia. Circulating maternal sex hormones cause gynaecomastia in a small number of male neonates. This usually resolves within **six months**.
- Pubertal gynaecomastia. It occurs in up to **70%** of normal pubertal boys and is often asymptomatic. It usually resolves within **2 years** when adult testosterone levels are reached.
- Senile gynaecomastia. The increased prevalence of gynaecomastia with age is related to the **reduction** of testicular function.

Secondary gynaecomastia

- Reduced production of **testosterone as after orchidectomy for prostatic** cancer, and **testicular atrophy** after **viral orchitis** and leprosy.
- High levels of oestrogen caused by feminising testicular tumour and suprarenal tumours.
- Failure to metabolize oestrogens in cases of chronic liver disease.
- Drugs as oestrogen preparations, and long term administration of **cimetidine, digoxin, spironolactone, phenothiazines, and cannabis**.
- Ectopic hormonal production in bronchogenic carcinoma.



Clinical Picture

- The usual complaint is breast enlargement which may be unilateral or bilateral, and with or without tenderness.
- Examination reveals a subareolar mass (a disc of tissue) which is soft and mobile. Any eccentricity, induration, or fixation should raise the suspicion of malignancy (this is not a possibility in children and adolescents). Examination should include the **abdomen and testes**.



Investigations

These are not required for neonatal and adolescent cases.

1. **Liver function** tests, and **hormone assays** in appropriate cases.
2. **Biopsy** in cases suspected of malignancy.



Treatment

- Most cases require no treatment. **Reassurance** is all that is required for **physiological** gynaecomastia. The neonatal and adolescent types usually resolve spontaneously.
- **Secondary gynaecomastia** will often improve following treatment of the underlying condition or withdrawal of the responsible medication.
- **Persistent gynaecomastia** causing embarrassment is treated **surgically** by **subcutaneous mastectomy** or by **liposuction**.

Carcinoma of the male breast

- It is **rare**.
- Because of the deficiency of breast tissue it **rapidly becomes attached** to the skin and chest wall, and ulceration is common.
- The main **differential diagnosis** is from **gynaecomastia**.
- Staging and treatment are similar to that for carcinoma of the female breast. The usual operation is **modified radical mastectomy**. **Chemotherapy** and tamoxifen are usually indicated postoperatively.
- Prognosis is **worse** than that of female breast cancer.

Thyroid Gland

Thyroglossal Cyst



Pathophysiology

- It is a midline **tubulodermoid cyst** arising in thyroglossal duct remnant.
- The thyroglossal duct has a **variable relationship** with the **hyoid bone**; behind, in front or passing through its body.
- The cyst may occur at any level from the foramen caecum of tongue to the suprasternal notch, usually in the midline **just below hyoid bone** except at the level of the thyroid cartilage, where it is displaced **usually to the left**.



Clinical Picture

The cyst may present at any age, most common in **childhood**.

1. **Painless cystic** swelling moving up and down on swallowing & on protrusion of the tongue (due to its relation to hyoid bone).
2. There may be a **palpable track** extending from the hyoid bone upwards towards the tongue. Thyroglossal cyst.
3. The wall of the cyst is rich in **lymphatics** which may communicate with the cervical lymph nodes, so **infection** may be the presenting symptom and may even lead to **fistula formation** when an infected thyroglossal cyst ruptures or is incised.



Differential Diagnosis

Dermoid Cyst - Lymph Nodes



Treatment

- Surgical excision of the cyst and its associated tract then removal of the middle third of the body of the hyoid bone (**Sistrunk's operation**).
- If the excision is incomplete, **recurrence** in the form of thyroglossal cyst or fistula may result.
- In patients presenting by an infected cyst, treatment is by **antibiotics and drainage** of the abscess.

Thyroglossal Fistula



Aetiology

This is an **acquired** fistula that is caused either by:

- **Infection** of a thyroglossal cyst leading to rupture
- **Inadequate** removal of the cyst.



Pathophysiology

The track is lined by columnar epithelium, discharges mucus, and presents with recurrent inflammation.



Clinical Picture

The **opening** of the fistula is near the midline on the **left side** with a **crescentic** fold.

A **track** can usually be **palpated** extending from this opening upwards to the hyoid bone.



Treatment

Is by **excision** of the fistula together with the central part of the hyoid bone. If there is an **upward extension** beyond the hyoid bone it should be followed up even to the foramen caecum of the tongue.

Lingual Thyroid



Pathophysiology

Failure of descent of the thyroid can lead to an intralingual development of the gland.



Clinical Picture

patients may present with a **tongue swelling** with **obstructive manifestations** i.e. dysphagia, difficult breathing or change in the quality of voice or even haemorrhage.



Investigations

Thyroid scans will establish the absence of thyroid tissue in the normal site in the neck.



Treatment

Options include full replacement with **L-thyroxine** to induce reduction in size, surgical extirpation or radioactive-iodine ablation.

Goitre Classification

This term means generalized thyroid enlargement. Based on the aetiological and clinical picture goiter may be classified into:-

1. Inflammatory goitre (Thyroiditis):

- Acute bacterial (rare).
- Subacute thyroiditis (De Quervain's disease).
- Chronic, e.g. tuberculosis or syphilis (rare).
- Autoimmune goitre as Hashimoto's thyroiditis or Riedel's thyroiditis.

2. Toxic goitre:

Means thyroid enlargement accompanied by the clinical picture of hyperthyroidism.

3. Neoplastic goitre:

May be benign or malignant.

4. Simple goitre:

Means thyroid enlargement, not accompanied by inflammatory, toxic or neoplastic criteria. This may be: Simple physiological goitre - Simple colloid goitre - Simple nodular goitre.

Thyroiditis

Subacute thyroiditis

- Also: granulomatous or De Quervain's thyroiditis.
- Aetiology is controversial, most probably a viral infection.
- It has a moderately **abrupt onset**, often following an **upper respiratory infection**. The thyroid becomes congested, swollen with a firm irregular pattern and slightly tender. It is accompanied by **neck pain** with **fever** and malaise.
- Elevation of **ESR** with a normal or depressed leucocytic count is a feature. **Iodine-uptake** by the gland is depressed in the presence of a slight elevation of serum T4.
- It is characterized by **remissions & exacerbations** over a period of up to few months, but usually it is self-limited. A **rapid response to oral prednisone** is diagnostic, and a course of the drug may be required for a few weeks. In severe cases, anti-inflammatory drugs are beneficial.

Riedel's Thyroiditis

- This is a rare condition, accounting for 0.5% of goitres.
- It presents as a **hard woody** transformation of thyroid tissue due to **extensive fibrosis** which extends even beyond the gland. It may occur in **association** with retro-peritoneal fibrosis. Some authors consider this disease as an autoimmune disorder, others consider it as a collagen disease.
- Hypothyroidism is usually present. The differentiation from anaplastic carcinoma may mandate an **open biopsy** where a wedge of the isthmus is removed to free the trachea.

Autoimmune thyroiditis (Hashimoto's thyroiditis)

It is the most common form of thyroiditis, usually affects **females at menopause**.

Aetiology Probably due to the presence of **antibodies to thyroid antigens**.

Onset:

It exhibits a variable onset being insidious and asymptomatic in some cases or sudden and painful in others.

Pathophysiology

- There is **infiltration** of the thyroid gland by **lymphocytes and plasma cells**.
- Eventually the **acini are replaced** by this infiltrate leading to the development of myxoedema.

Clinical Picture

The goitre is usually **lobulated** (flat-topped nodularity), diffuse or localized to one lobe.

It may be large or small, soft, rubbery or firm in consistency.

Mild hyperthyroidism may be present initially but **hypothyroidism is inevitable**.



Investigations

1-Serum titres of **antimicrosomal** and **anti-thyroglobulin** antibodies are elevated.

2-fine needle or true-cut needle biopsy (may be required) to differentiate it from nodular goitre or carcinoma.



Treatment

- **Includes full** replacement dosage of thyroxine:
- **Surgical treatment** may be needed for: -large-sized goitres .
- Suspected development of **lymphoma** which is occasionally associated with Hashimoto's goitre or develops later in the disease.

Simple Goitre

Endemic or Sporadic

This term is applied to thyroid enlargement **not** belonging to the inflammatory, thyrotoxic or neoplastic groups.



Aetiology

Results from stimulation of the thyroid gland by **elevated levels of TSH**, as a sequence of an **absolute or relative** decrease in the circulating thyroid hormones.

1. Iodine deficiency:

- This may be absolute or relative. Normal daily requirements of iodine is **100-125 ug**.
 - In Egypt oasis areas are famous for endemic goitre.
 - **Relative** deficiency of iodine occurs during periods of stress:
- Females during the **menarche**, **pregnancy** and **lactation** are susceptible to this problem.

2. Enzymatic deficiency:

This is responsible for many cases of **sporadic** goitre.

- Often being associated with a **positive family history**, reflecting an underlying genetic defect.
- **(P)endred's syndrome**; is an example, being due to the deficiency of **(P)eroxidase** enzyme which transforms inorganic iodides to organic iodine.
- The child has goitre, deafness and mutism. sometimes several brothers or sisters are affected
- The patient is liable to get recurrent goitre after surgery.
- Deficiency of **dehalogenase** enzyme is also responsible for sporadic goitre.

3. Goitrogens:

Intake results ultimately in goitre formation. These include **thiocyanates** in **cabbages**, drugs as **para-amino salicylic acid** and **antithyroid** drugs.

Stages of goitre formation:

1. Diffuse hyperplastic (physiological) goitre:

Deficient synthesis of thyroid hormone leads to persistent **TSH elevation** that produces diffuse **homogenous hyperplasia**;

- All follicles are active with uniform iodine uptake. and is reversible on cessation of its stimulus i.e. increased TSH.



- Other factors have been implicated in the aetiology of nodular goitre, e.g. immunoglobulins. These by acting on oversensitive cells and/or receptors produce a nodular goitre with marked heterogeneity of structure & function between adjacent follicles.
- The condition appears early in endemic regions but may be delayed to the third decade or later in sporadic cases.

-Treatment (of diffuse hyperplastic goitre):

It is **usually reversible** by the use of **L-thyroxine**, usually 0.2 mg/day (**200 micr-gram**) for several months to be **tapered** to 0.1 mg/day for several years.

The patient is reassured and is advised for

2. Simple colloid goitre:

- If the iodine requirements are **satisfied** by large doses of iodine.
- The follicles undergo **involution** or even **hyperinvolution** where the follicles are distended with colloid and are lined by flattened epithelium.

Clinically:

The gland is enlarged, the surface is **smooth** & its consistency is **soft**. The cut surface of the gland has a glistening surface as the follicles are **full of colloid**.

3.Stage of Nodular goitre :

- **Repeated fluctuations** in the level of TSH due to repeated cycles of stress & strain produce heterogeneity of the gland, with areas of active follicles & others with inactive follicles>> **recurrent** hyperplasia and hypervascularity.
- **Haemorrhages** may occur producing **central necrosis** in the lobules. **most nodules are inactive**, the active follicles being present in the internodular tissue.



Clinical Picture (of simple nodular goitre)



Symptoms

1. Cosmetic deformity: The patient's main complaint is usually the cosmetic deformity or some respiratory obstruction.

2. Sudden enlargement: with the appearance of **pain** & tenderness usually results from haemorrhage into a nodule.



Signs

1. The thyroid gland is **firm** in consistently with a **nodular** surface.
2. The nodules are smooth, firm & **painless**.
3. Calcification results in **hardness** & irregularity simulating malignancy.



Complications (of simple nodular goitre)

1. Tracheal obstruction by compression:

- The trachea may be compressed on either side & becomes transformed into anteroposterior slit (**scabbard trachea**).
- it may be compressed anteroposteriorly, or it may be **displaced** to one side by marked unilateral enlargement.
- In long-standing goitre, the tracheal rings undergo chondromalacia and the tracheal rings may collapse post-operatively.

2. Secondary thyrotoxicosis: may occur in up to **30%** of cases.

3. Malignancy: Occasionally **follicular** carcinoma may develop. The incidence is about **3%**.

4. Cyst formation.

5. Haemorrhage: into a nodule.

6. Calcification: may occur in longstanding cases.

7. Retrosternal extension.



Investigations

The patient is euthyroid.

1. **Thyroid functions:** to exclude mildly toxic nodular goitre.
2. **Thyroid antibodies:** to exclude Hashimoto's disease.
3. **FNAC:** should be performed, if a nodule is suspicious of malignancy.



Prevention

- The use of **iodized table salt** (potassium iodide 1 part: 10.000) has proved successful for prophylaxis in endemic areas.
- Regular **follow-up**.



Treatment

- Most authorities agree upon the **irreversibility** of multinodular goitre, thus surgical excision (partial thyroidectomy) is indicated for:
 1. Evidence of **compression** mainly on the trachea.
 2. **Cosmetic** reasons.
- Surgery includes removing the nodular parts leaving an equivalent of **8 gm** of relatively normal thyroid tissue (size of a normal lobe) on each side if feasible.
- Sometimes the distribution of the pathology requires removal of a whole lobe & subtotal excision of the other lobe.
- **Post-operatively** suppressive doses of L-thyroxine 0.1-0.2 mg/day to avoid recurrence in the remainder of thyroid tissue.



Surgery for multinodular goitre should not be advised below the age of 25 unless very much indicated as it may be followed by recurrence.

Retrosternal Goitre



Pathophysiology

Retrosternal goitre is classified into 3 types:

1. **Plunging goitres:** which **rise with deglutition** & then descend again through the thoracic inlet.

2. **Mediastinal goitres:** The **whole goitre** lies in the chest, but are **connected** with the thyroid by a relatively narrow band of tissue and derive their blood supply from the thyroid vessels.

3. **Intrathoracic goitres:** The **whole goitre** lies in the chest, completely **separate** from the main gland.

They probably arise from congenitally misplaced thyroid tissue, they have an **independent blood supply** from mediastinal vessels (not carotid).



Clinical Picture



Incidence

A retrosternal goiter is more common in **men**, particularly **short-necked** individuals.



Symptoms

Because it often remains symptomless for years, the patient usually presents after **middle age**.

1. **Dyspnea:** the goitre displaces & compresses the trachea, which is **worse at night**, and is often **spasmodic and positional** being aggravated by any posture that reduces the thoracic inlet, such as lying down or flexion of



Retrosternal goitres usually arise in normally placed thyroid glands. The sternohyoid & sternothyroid muscles prevent forward expansion, & assisted by gravity & the negative intrathoracic pressure, they direct the swelling into the mediastinum.

the neck, so that many patients prefer to **spend the night in a chair**.

Some patients are diagnosed as asthmatics.

2. Dysphagia: Sometimes there is dysphagia.

Signs:

1. Inspection: superficial veins of the upper part of the chest are dilated because The innominate veins may be obstructed.

There may be cyanosis of the face.

2. Palpation: of the neck may reveal an enlarged thyroid gland.

3. Percussion: of the sternum may reveal retrosternal dullness.



Investigations

1. Plain CXR: shows a shadow in the **superior mediastinum**.

Calcification often occurs in the walls of the goitre, and may be demonstrated radiologically.

2. 99mTc scan (Thyroid scan): can reveal the retrosternal extension.

3. CT scan: of the thorax will reveal the exact level of the retrosternal extension and its anatomic relations.



Treatment

- Thyroidectomy is the only line of treatment.
- Resection is almost always possible via the **cervical approach**, rarely a **median sternotomy** is required. **Devascularization** is done via the neck from which the restrostemal portion derives its blood-supply.

Toxic Goitre

(thyrotoxicosis = hyperthyroidism)

Common types:

1. Diffuse toxic goitre (primary toxic/Graves' disease): 76%.

2. Toxic nodular goitre (secondary toxic/Plummer's disease): 14%.

3. Toxic nodule: 5%.

Rare causes of hyperthyroidism:

1. Thyrotoxicosis factitia:

Due to excess intake of **L-thyroxine** (>0.3 mg per day).

2. Jod-Basedow thyrotoxicosis:

Occur in cases of hyperplastic endemic goitre upon the administration of large doses of **iodine**, the condition is temporary and very rarely permanent.

3. Subacute thyroiditis (De Quervain's disease):

Mild manifestations of thyrotoxicosis may occur due to liberation of hormones from destroyed tissue.

4. Hashitoxicosis:

About 5% of patients with Hashimoto's disease may have hyperthyroidism in the early stages of the disease.

5. A large mass of secondary carcinoma: May be functioning.

Most but not all symptoms & signs of thyrotoxicosis are due to excess thyroid hormones. Many systems are vulnerable to these hormones.



- There may be multiple extrasystoles, paroxysmal atrial tachycardia, paroxysmal atrial fibrillation or persistent atrial fibrillation not responding to digoxin.
- In severe cases proximal myopathy & muscle atrophy may occur. The proximal muscles as the shoulder & plevic girdle muscles are particularly affected.

6. Neonatal thyrotoxicosis:

-May occur in newborns of **thyrotoxic mothers** or of mothers with history of thyrotoxicosis, due to transmission of thyroid antibodies across the placenta. -The condition subsides in 3-4 weeks upon decline of the titre of the antibodies in the baby's serum.

7. TSH secreting adenoma: Of the pituitary gland.



Pathophysiology



Pathogenesis

1. Diffuse Toxic Goitre

An excess of thyroid hormones produces a state of hypermetabolism that affects the whole body systems. The **nervous, metabolic & eye** manifestations are more marked than the cardiovascular. Some of the manifestations of Graves' disease are not due to hyperthyroidism, e.g. the exophthalmos & pretibial myxoedema.



Microscopic

- There is **diffuse enlargement** of the whole thyroid gland.
- There is **columnar proliferation** of the epithelial lining of the acini which may be arranged in several layers. The cells are columnar, & **full of granules**.
- The **acini** contain **less vacuolated colloid** or are devoid of it.
- There is **marked increase in vascularity** making surgery, if indicated, difficult.
- Extensive lymphocytic **infiltration** is present.

2. Toxic Nodular Goitre

(Plummer's disease)

- This occurs on top of a long-standing simple nodular goitre, so affects **middle** aged and **elderly** persons.
- It is very rarely, if ever, accompanied by extra-thyroidal manifestations, e.g. advanced exophthalmos.
- It is usually the **internodular** tissue that becomes hyperactive, but occasionally one or more nodules become the site of hyperactivity.
- The onset is usually **insidious** & may present with **cardiovascular** manifestations.

3. Autonomous Toxic Nodule

- Toxicity is due to a **solitary, hyperactive**, autonomous nodule (**not** due to thyroid antibodies).
- The toxic nodule produces **suppression of TSH** secretion with subsequent suppression of the remainder of the thyroid gland which becomes almost atrophic as **evident on isotope scanning**.



Clinical Picture

(Of all types of thyrotoxicosis)

1. Metabolic:

There is **loss of weight** in spite of **increased appetite**. The patient complains of excessive sweating and intolerance to hot weather. The **hands** of the patient are **warm** due to vasodilation and are **moist**.

• Diffuse Toxic Goitre is due to presence of thyroid stimulating antibodies which combine with TSH receptors in the thyroid gland cells leading to the release of cAMP. This will lead to activation & increased production of T4.

• This disease affects mainly young females, is usually of abrupt onset & is characterized by remissions & exacerbations.



2. Nervous system:

- **Symptoms:** Nervousness, bad temper, insomnia or bad dreams.
- **Signs:** The patient looks tense & irritable. There may be tremors in the outstretched hands. Reflexes (as knee jerk) are exaggerated.

3. Cardiovascular system:

- **Symptoms:** The patient complains of **palpitation** sometimes even at rest. There may be anginal pain. If there is heart failure, the patient complains of orthopnea, pain in the right hypochondrium and bilateral oedema of the lower limbs.
- **Signs:**
 - (a) Tachycardia with a sleeping pulse more than **90/minute**.
 - (b) Water hammer pulse is due to high systolic (due to increased cardiac output) and low diastolic blood pressure (due to decreased peripheral resistance).
 - (c) Arrhythmias: All sorts of cardiac arrhythmias except heart block may occur.

4. Muscular: weakness with rapid exhaustion after minor effort.

5. Skin: Patchy pigmentation or vitiligo may occur. **Pretibial myxoedema** presents as multiple orange yellow patches of thickening of the skin over the shin of the tibia. They are due to deposition of **mucin** like substances.

6. Gastrointestinal tract: there may be diarrhoea.

7. Endocrine system: Menstrual abnormalities may occur in females as menorrhagia. There may be abnormal libido, gynaecomastia or impotence in males.

8. Urinary system: There may be polyuria or glycosuria.

9. The eyes:

- Exophthalmos:

(a) **True Exophthalmus:**

- means actual protrusion of the eyeball, is due to **deposition** of fluid and round cell infiltrate in the retrobulbar tissues.
- It is characteristic of **Graves' disease**. - The condition is aggravated by **ophthalmic vein compression, leading to lidoedema, conjunctival injection and chemosis**.

(b) **False Exophthalmus:**

- is due to **retraction** of the upper eyelids, and accompanies both diffuse and nodular toxic goitres.
- It is due to contraction of the levator palpebrae supenons (**Muller's muscle**) . - The high level of thyroid hormones **sensitizes** this unstriated muscle to the effect of circulating catecholamines.
- Alpha-adrenergic blocking drugs as guanethidine eye drops improve false exophthalmos.



- Weakness of extra-ocular muscles results in diplopia.

- In severe cases papilloedema, corneal ulceration and optic nerve neuropathy may occur.

- The severe progressive form is known as malignant exophthalmos.

- It is postulated that true exophthalmos is an autoimmune disease due to thyroid stimulating antibodies affecting the ocular muscles.

- False exophthalmos usually disappears when the hyperthyroidism is treated.

SEE OUR PRACTICAL BOOK FOR METHODS
TO DETECT TRUE EXOPHTHALMOS.

• Eye signs:

Eye signs other than exophthalmos, include:

1. **Dalrymple's sign:** Appearance of a rim of sclera between the upper eyelid and the cornea.
2. **Stellwag's sign:** **Staring** look with infrequent blinking.
3. **Von Graefe's sign:** The upper eye lid lags behind the moving eyeball as the patient looks down without moving the head.
4. **Joffroy's sign:** Lack of forehead **wrinkling** on looking upwards without moving the head.
4. **Moebius's sign:** Defective **convergence** due to muscle paresis.

1. Local signs of toxic goitre in the neck:

• In diffuse toxic goitre,

- the thyroid gland is diffusely enlarged.
- The gland is soft .
- A thrill and bruit may be detected at the upper pole over the superior thyroid arteries.
- The surface of the gland is smooth and the overlying skin may be warm.
- The gland may show expansible pulsations.

• In nodular toxic goitre,

- the thyroid gland is **firm** in consistency with a **nodular** surface. The size of the gland may be large.

Clinical examination provides adequate basis for the diagnosis in most cases.

Thyrotoxicosis should be suspected in:

- Children with **growth spurt**, behavioural problems or **myopathy**.
- Elderly persons with unexplained **tachycardia** or arrhythmia (apathetic hyperthyroidism).
- Unexplained **diarrhoea**.
- Unexplained **weight loss**.
- Resistant **heart failure** (marked thyrotoxicosis).

**CLINICAL DIFFERENCES BETWEEN THE TWO
MAIN TYPES OF THYROTOXICOSIS**

	Graves' disease	Toxic nodular goitre
Age	Young	Elderly
Onset	Abrupt	Gradual
Course	Exacerbations & remissions	Steady
Nervous symptoms	+++	
Metabolic manifestations	+++	+
Cardiovascular manifestations	+	+++
Eye signs	+++ exophthalmos	Usually no exophthalmos
Thyroid	Diffuse enlargement soft and vascular	Multiple or solitary nodules



Investigations

1. Thyroid Function Tests: T3, T4, TSH & 123I uptake test to confirm the diagnosis. T3 thyrotoxicosis is identified by elevated free T3.
2. Toxic adenoma shows as a hot nodule on 123I scanning with suppression of the uptake of the surrounding thyroid tissue.



Treatment

The treatment of thyrotoxicosis comprises three options:

Medical - Radioactive iodine - Surgery



Medical

Indications:

1. Primary thyrotoxicosis.
2. Mild thyrotoxicosis.
3. Children and young patients because surgery may affect their growth.
4. Pre-operative preparation.
5. Post-operative recurrence.
6. Refusal of surgery.
7. Small gland.

Advantages:

- Avoiding surgical risks.
- Avoiding the possible hazards of radioactive materials.

Drawbacks:

1. There is no way to accurately predict which patient is liable to pass into remission.
2. High relapse rate which amounts to 60% within 2 years from stopping treatment.
3. Further enlargement of the gland may occur.
4. Adverse effects of the drugs: Thiouracil & carbimazole may cause gastrointestinal upset, rashes, arthralgia & reversible bone marrow depression leading to agranulocytosis.

Types of drugs used:

• Carbimazole:

-Action: It blocks iodine **binding** to tyrosine and decreases antibody titres. It is the most commonly used drug in Egypt.

-Dose: The initial dose is **10 mg** three times daily with a maximum dose of **60 mg** daily.

-Effectiveness:

There is a **latent** interval of **7-14 days** before clinical improvement takes place.

- When the euthyroid state is reached, the dose is gradually reduced to 5 mg three times a day, and is maintained for 12-18 months.

• Propylthiouracil

-Action: It blocks iodine **binding** to tyrosine & prevents **peripheral conversion** of T4 to the more active T3.

-Dose: **100 mg t.d.s.**

- **Potassium perchlorate:**

It competitively inhibits iodide **uptake** by the thyroid.

- **Propranolol or other beta-adrenergic blockers:**

-Action: inhibit the **cardiogenic effects** of thyroid hormones by beta-blockade.

- **Iodides:**

Action:

- They reduce the **effect of TSH** on the thyroid gland & inhibit **iodine binding**.

-They also reduce the **vascularity** of the thyroid gland & lead to storage of colloid within the acini.

-Their effect is only **temporary** & so they cannot be used for long term control. Their main use is for **preoperative** preparation.



Non selective beta-adrenergic blockers e.g., propranolol must not be used in asthmatic patients.



Radioactive iodine:

Mechanism of action:

Radioactive iodine destroys the thyroid cells thus reducing the mass of functioning thyroid tissue.

Dose:

-The suggested dose is **160 uCi** per **1 gm** of thyroid tissue.

-The response is usually slow, improvement is expected after **8-12 weeks**, if not a second dose or several doses may be required, which is the case in 20-30% of these patients.

Possible side effects:

Radioactive iodine treatment has long been blamed for causing :

-**genetic** damage, **leukaemia**.

-damage to the **fetus** if administered in early pregnancy.

- increasing the incidence of thyroid **carcinoma** in adults 10-15 years after its administration.

NB: so the minimum age for its use in many centers is now 25 years.

Indications:

1. Diffuse toxic goitre.
2. Thyrocardiac patients.
3. Refusal of surgery.
4. Recurrence after surgery.

Advantages:

- Avoidance of surgery or prolonged medical therapy.

Disadvantages:

Progressive destruction of the gland resulting in thyroid insufficiency (Iatrogenic Hypothyroidism) in up to **75- 80%** of cases after **10 years** and so an indefinite follow-up is mandatory.

NB: Inadequate for the treatment of secondary toxic goitre.as most nodules have low iodine uptake(cold).



Surgical

The patient should be in a euthyroid status before the operation,
NB: Otherwise, he may develop a **post-operative thyroid crisis**.

A- Preparation :

-by carbimazole and propranolol is usually satisfactory. Some surgeons recommend prescribing iodides (**Lugol's iodine 10 drops t.d.s.**) for **10 days** preoperatively to diminish the **vascularity** of the gland and render it more firm.

-A **sleeping pulse of 80/min** is considered as effective preoperative preparation.

Advantage:

1. Rapid cure.
2. High rate of success.

Indications:

1. Secondary toxic goitre.
2. Severe primary thyrotoxicosis.
3. Retrosternal toxic goitre.
4. Failure of medical treatment.
5. Occurrence of side effects due to medical treatment.

Drawbacks:

1. Morbidity & mortality are negligible in experienced hands.
2. Recurrence rate less than **5%**.
3. Thyroid insufficiency at an incidence of **20-45%**.
4. Parathyroid insufficiency in less than **0.5%** but is a definite risk even in experienced hands because of the high incidence of anatomical variation of the site of the parathyroid glands.

Special problems in management

Thyrotoxicosis With Pregnancy

- Radio-iodine is absolutely contraindicated, because destruction of the fetal thyroid would result.
- Antithyroid drugs at conventional doses would result in foetal hypothyroidism with the development of goitre that may obstruct the airway. -Minimum doses antithyroid drugs, supported by B-blockers, reduce this risk.
- The dose of an antithyroid drug is adjusted according to **serum level of free T4**.
- Surgery after a short course of antithyroid drugs and **propranolol** proved to be **safe** during the second or 3rd trimester.
- During **lactation, propyl-thiouracil** is recommended as it is excreted in a harmless very low concentration in milk.

Thyrotoxicosis In Children

- Radioiodine is **contraindicated** for children.
- Conventional surgery is either followed by a high recurrence rate, due to high activity of the cells in the young or followed by a hypothyroid state which affects later growth.
- Preferred to tide them over by **antithyroid drugs till late teens**.

The Thyrocardiac Patient

- The cardiac condition takes priority in management.
- Thyroidectomy is ideal after control of the cardiac status.
- If it is not permissible, radioiodine is used followed by antithyroid drugs until the effect of the former appears (6 weeks).

Proptosis Of Recent Onset

- It is not preferable to **terminate** the toxic status **abruptly** by surgery or radio-iodine for fear of the theoretical risk of progressing to malignant exophthalmos.
- **Antithyroid drugs** are used until the proptosis has been stable for **6 months** after which total thyroidectomy is recommended.

Hypothyroidism



Aetiology

1. **Failure of development** of the thyroid gland.
2. **Dyschromogenesis** means congenital deficiency of one of the enzymes necessary for the formation of thyroid hormones, e.g. peroxidase enzyme.
3. **Failure of secretion** of TSH by the anterior pituitary, e.g. in Sheehan's syndrome.
4. **Primary myxoedema** is supposed to be an **autoimmune** disorder. It may be associated with Addison's disease.
5. **Iatrogenic** following subtotal thyroidectomy or radio-active iodine treatment particularly for toxic goitre.
6. In late stages of **Hashimoto's** thyroiditis.



Clinical Picture

Cretinism:

All the parameters of normal development are impaired.

The infant does not grow properly:

-he is **stunted**, delayed **teething** or **walking**.

- The child has an **apathetic look**.

with depressed nose, thick lips and thick protruding tongue.

-The abdomen is distended (**potbelly**) and there is usually an **umbilical hernia**.

Myxoedema:

- The symptoms of myxoedema are nearly opposite to thyrotoxicosis.

The patient complains of lethargy, weakness, loss of appetite, putting on weight and intolerance to cold weather.



Signs

- The patient looks apathetic with prolonged reaction time.
- The face is puffy with supraclavicular pads of fat.
- The skin and hairs are dry.
- The hands are cold.
- There is bradycardia with a low volume pulse.
- The voice of the patient is coarse. -Pericardial effusion, carpal tunnel syndrome, deafness and ataxia may be present.



Complications

The danger of myxoedema is that the diagnosis may be missed and the condition may progress to **coronary thrombosis** due to **hypercholesterolaemia**.

-A myxoedematous patient if subjected to a stressful situation may develop coma (**Myxodema coma**)



Investigations

- T3 and T4 levels are low.
- **TSH** is markedly **raised**.



Treatment

-**Replacement** by **L. thyroxine 0.2-0.3 mg/day** for adults is satisfactory.



Very early treatment of cretin infants may save these patients, otherwise the changes are irreversible.

Thyroid Neoplasms

Classification

The thyroid gland contains follicular and parafollicular (C) cells. Tumours of the thyroid gland can be classified as follows:

1. Tumours arising from follicular epithelium:

- Benign → Follicular adenoma
- Malignant
 - Differentiated
 - Papillary carcinoma
 - Follicular carcinoma
 - Mixed carcinoma
 - Undifferentiated → Anaplastic carcinoma

2. Tumours from parafollicular epithelium: Medullary carcinoma.

3. Tumours from lymphoid elements: Malignant lymphoma.

4. Rarely the thyroid gland is infiltrated by metastatic deposits or by local infiltration from a nearby lesion.

Thyroid Cancer

DIFFERENCES BETWEEN THE COMMONEST THREE TYPES			
	Papillary	Follicular	Anaplastic
Incidence	60%- 70%	%17	1%-5%
Age	May occur in children and young adults	Middle age	Elderly
Sex (F:M)	3.5:1	2:1	1 :1.3
Microscopic picture	Papillary projections formed of connective tissue covered by a single layer of epithelial cells with pale empty nuclei. Laminated calcified bodies (Psammoma bodies) are often present.	Thyroid follicles with a variable degree of differentiation, Solid sheets may be present. Diagnosis depends on finding capsular or vascular invasion or on detectin metastases.	May take the form of spindle cell or large cell type.
Multiplicity	Up to 80%. May be due to multicentricity or intrathyroid lymphatic spread.	Rare	
Spread	Mainly lymphatic. Blood spread may occur with tumours that extend outside the thyroid.	Mainly blood spread	Mainly direct invasion. Lymphatic or blood spread.
10-year survival	90%	Encapsulated:97% Invasive:70%	Most patients die within 1-2 years.

Predisposing Factors

1. External irradiation of the head and neck region in children which was previously used for treatment of haemangiomas and thymic gland enlargement.
2. Follicular carcinoma is the commonest type to develop in endemic goitrous areas, possibly due to continuous stimulation of the thyroid gland by TSH.
3. A genetic element may be present as the lesion is more common in certain families.
4. Malignant lymphoma may occur on top of Hashimoto's disease.



If the histopathology contains both papillary & follicular elements it is managed as papillary. Features of papillary carcinoma considered high risk & associated with worse prognosis include male gender, primary tumor size >4 cm, gross local invasion & extrathyroidal extension, age >45 years, certain histologic subtypes (specifically, the tall cell variant), lymphovascular invasion, or known metastatic disease.

Medullary Carcinoma



- It accounts for 6% of malignant neoplasms.
- It spreads to lymph nodes in 50-60% or gives rise to blood borne metastases.
- The tumour arises from the parafollicular cells & is, therefore, considered a part of the apudomas. It secretes calcitonin.
- The tumour may occur alone, or it may be associated with hyperparathyroidism & pheochromocytoma constituting (MEN IIa).
- The tumour may be familial and in this case it is more common in children & young adults. Genetic testing (RET proto-oncogene) is recommended.



Microscopic

- The tumour is composed of sheets of neoplastic cells set in a hyaline stroma which may contain amyloid material. Mitotic rate of growth is usually low.



Clinical Picture



Symptoms

1. Thyroid swelling: which is enlarging **rapidly** over some months. **Sometimes**, the swelling is **painful** and the pain may be **referred** to the ear (referred along the auricular 'Arnold' branch of vagus).
2. Compression manifestations: If the lesion is advanced, the patient may complain of **dyspnea or change of voice** due to involvement of the recurrent laryngeal nerve.



Signs

1. A lump, firm to **hard** in consistency, occupying a part or the whole of the thyroid gland. There may be some **restricted mobility** of the thyroid.
2. In anaplastic tumours, the rate of growth will be more **rapid** and there will be **early infiltration** of the surrounding structures as the trachea, recurrent laryngeal nerve or the muscles.
3. The examiner should look for **lymphatic** or **blood borne metastases**, the latter occur usually in the bones particularly the **spine, skull or neck of femur**, in the lungs or in the brain.
4. Some low grade differentiated tumours present exactly as benign lesions. Any solitary nodule of the thyroid particularly in young males is suspicious.
5. Medullary carcinoma may produce **diarrhoea in 30%** of cases due to production of **5 hydroxytryptamine or prostaglandins**.
6. Occult carcinoma: some patients may present, by metastatic **lymph nodes** in the jugular chain while the **primary papillary carcinoma is not palpable**.

Investigations:

A- To diagnose:

1. Ultrasound:

can prove whether the lesion is **cystic, solid or cystic with solid projection**.

The following ultrasound features are suggestive of malignancy in a thyroid nodule:

- | | |
|-------------------------|-----------------------------|
| -Hypoechoic. | -Uneven borders (irregular) |
| -Increased vascularity. | -Microcalcification. |
| -Size more than 1 cm. | -Metastatic lymph nodes. |

2. Thyroid isotope scanning:

- is of **limited** diagnostic significance. Malignant lesions usually appear as cold areas in the scan.

NB: It should be remembered that many benign lesions as cysts or haemorrhage in a cyst or degenerative nodules or thyroiditis appear also as cold areas.

3. Aspiration of a Cyst:

- a. A benign cyst is characterized by:
 - i. The aspirate is **clear**.
 - ii. Complete **disappearance** of the cyst.
 - iii. No **reaccumulation** of fluid.
 - iv. **Cytological** examination is free.
- b. A cystic malignant lesion is suspected when:
 - i. The aspirate is **haemorrhagic**.
 - ii. A **residual** lump is present.
 - iii. Rapid **reaccumulation** of fluid in the cyst.
 - iv. **Cytology** may be **positive** for malignant cells.

4. Fine Needle Aspiration Cytology (FNAC):

- The role of this **simple, rapid & inexpensive** investigation is becoming increasingly reliable.
- An **experienced** pathologist can give an accurate result in about **90%**.
- In **follicular carcinoma** FNAC may not be conclusive as the diagnosis of this lesion depends upon demonstration of capsular or blood vessel invasion (not depending on cellular atypia).

5. Surgical Exploration: If the diagnosis is still in doubt, the best policy is to do surgical exploration of the thyroid and completely resect the affected lobe. It is recommended to do **frozen section**, so that complete definitive treatment is performed simultaneously.

B-For Staging:

6. Chest x-ray: to detect pulmonary deposits.

7. Indirect Laryngoscopy: for the mobility of the vocal cords.

C- For follow up:

8. Tumour Markers:

- Estimation of **serum calcitonin** is not a routine investigation but is raised in many patients with **medullary carcinoma**, (considered as a tumour marker)
- **Thyroglobulin** is a tumour marker for **differentiated** thyroid carcinoma.



Treatment

Differentiated Carcinoma:

A. Total thyroidectomy and post operative Replacement therapy is recommended because:

1. High incidence of **multicentric** lesions in papillary carcinoma.
2. Total thyroidectomy ablates all thyroid tissue & makes it **possible to detect metastases** by scanning & to treat them by radioactive iodine which is not possible in the presence of normal thyroid tissue.
 - **L-Thyroxine 0.2 mg/day** is prescribed to **suppress TSH** production as most differentiated carcinomas are TSH dependent.

B. Postoperatively a thyroid scan is performed to detect any residual lesion which if present is treated by an **ablative dose of radioactive iodine**.



Thyroid hormone suppression therapy:

Two stages:

1. initial TSH suppression:

below 0.1 mU/L.

2. lifelong TSH suppression:

at or below the lower limit of normal (0.1 to 0.5 mU/L)

Aim: to decrease recurrences & may improve survival.

Methodes:

- Oral levothyroxine is started at a dose of **1.4g/kg/day**.
- Adequacy of thyroid hormone replacement is assessed by measuring **TSH and FT4 6 to 12 weeks** after initiating therapy.

C- Management Of Lymph Node Metastases:

1. If there are no palpable metastatic deposits: **follow up** is needed or the central lymph nodes are excised specially in high risk group.
2. If there metastases in the posterior triangle: a **modified block dissection** is performed in which the **accessory nerve, internal jugular vein and sternomastoid muscle** are **preserved**.

D-Management Of Blood Borne Metastases:

- **Thyroxine is stopped for a few weeks** or substituted by triiodothyronine (to **raise TSH**) .
- Then **iodine scan** is performed> If there is evidence of **local recurrence** or metastases: **therapeutic doses of radioactive iodine are prescribed**.

Medullary Carcinoma:

This tumour **does not concentrate radioactive iodine** & is **not** under the control of **TSH**.

- Treatment is by **total thyroidectomy & resection of metastatic lymph nodes**.

1- **Total thyroidectomy alone** is only indicated for **MEN II** patients who have thyroid **nodules <5 mm & calcitonin levels <40 pg/L**.

2- **Otherwise**, treatment of both hereditary and sporadic MTC requires at least **total thyroidectomy with central neck compartment (level VI) lymph node dissection**.

3- **In patients with palpable primary tumors**: additional “dissection of **ipsilateral lateral compartment nodes** should be performed

Anaplastic Carcinoma:

- 1• the **majority** of these patients are **inoperable** when first seen & are considered a fatal outcome within months.
- 2• The usual treatment is **external irradiation** and **chemotherapy**.
- 3• If there is **tracheal compression**, the **isthmus is resected**.

Malignant Lymphoma:

Radiotherapy and chemotherapy.

Certain factors worsen the prognosis:

1. Age: Males above 40 and females above 50 years of age.
2. Size: of the lesion if more than 5 cm in diameter.
3. Presence of capsular or blood vessel invasion.

Solitary Thyroid Nodule



Aetiology

1. The nodule may be a **part** of **multinodular goitre**, other nodules are not clinically palpable (commonest).
2. **Toxic nodule**.
3. **Colloid** nodule.
4. **Adenoma**.
5. **Carcinoma**.



Investigations

1. Thyroid function tests and thyroid isotope scan: performed if there is suspicion of toxicity.

2. U/S: can differentiate between a cyst and a solid lesion.

3. Fine needle aspiration cytology (FNAC): This is a simple, fast, inexpensive investigation.

-depends upon cytological examination of nodules **0.5 cm or more**.

4. True cut needle biopsy:

-obtains a core of tissue for histological examination of nodules **2 cm or more** in diameter.

- It may cause haematoma.

5. Excision biopsy:

-The **surest** method of diagnosis is to do **lobectomy** (not enucleation) of the affected side.

- then perform **frozen section** histological examination in suspicious cases.

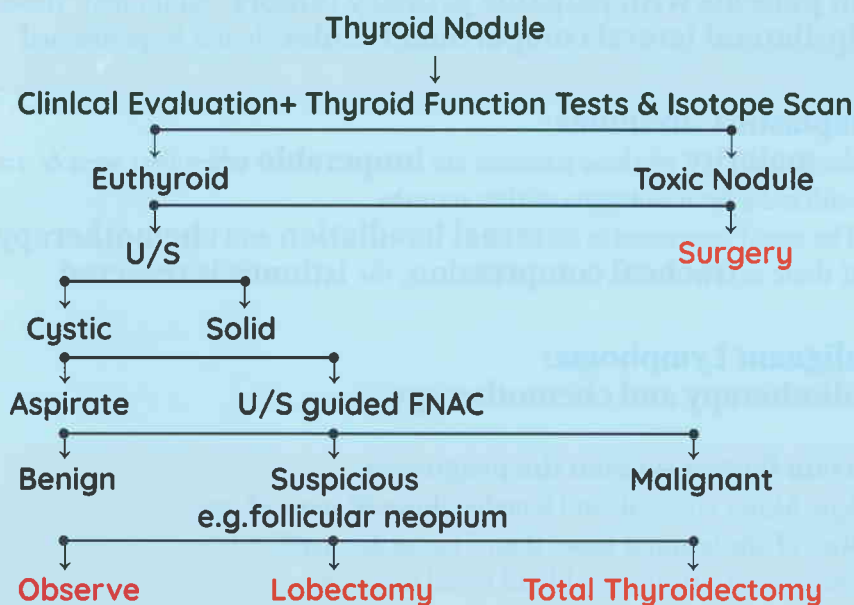
Thyroidectomy operations (see operative book)

especially **indications and complications**.

Findings that raise suspicion of malignancy!

1. History of irradiation.
2. Young or Elderly.
3. Recent onset and rapid growth.
4. Pain.
5. The nodule is hard, irregular, with limited mobility.
6. Local invasion or lymphatic or blood borne metastases.

Algorithm for the management of a solitary thyroid nodule



Development:

- The parathyroid glands arise from the branchial **pouches III and IV**.
- The **inferior** parathyroids are associated with the thymus gland and are derived from the **third** pouches.
- The **superior** glands are derived from the **fourth** pouches.

Number and position:

- In **80%** of cases there are **4** glands which are symmetrical in position on both sides but in the remaining cases there can be **more than 4** asymmetrically placed glands.

Blood supply:

Each gland is supplied by a branch from the **inferior thyroid artery** that enters at the hilum.

Sometimes the **superior** gland is supplied by the **superior thyroid artery**.



Difficulty is encountered in histological differentiation. The presence of a normal rim may suggest adenoma, the presence of locoregional invasion or metastases suggest cancer.

Parathyroid Glands Hyperparathyroidism

Types:

1. Primary hyperparathyroidism. There is **autonomous** production of the parathormone with loss of the feedback mechanism.
2. Secondary hyperparathyroidism. This type is due to compensatory hypersecretion of the parathormone secondary to **low serum calcium**.
3. Tertiary hyperparathyroidism. This starts as **secondary** hyperparathyroidism, but the **hyperplastic glands develop an autonomous function**.

Primary hyperparathyroidism



Incidence

- It is more in **females**, particularly in the fifth and sixth decades of life.
- The disease is sometimes **familial**, and is sometimes a part of the multiple endocrine neoplasia **MEN** either type I (Wermer's) or type II (Sipple).



Aetiology

1. A single adenoma [**92%**].
2. Multiple adenomata [**4%**].
3. Hyperplasia [**3%**].
4. Parathyroid carcinoma [**1%**].
5. Rarely ectopic PTH production by cancers especially of lung, kidney and bladder may produce manifestations of the disease.



Clinical Picture

Important Considerations:

- Hyperparathyroidism is more commonly detected due to measurement of serum calcium and phosphate in **screening** procedures of **asymptomatic patients**.
- In general **80%** of patients have renal involvement, and **35%** have skeletal involvement.



Symptoms

(Bones-Stones-Mood-GIT)

1. The earliest complaints are **muscle weakness, anorexia, nausea, constipation, polyuria and polydipsia**.

2. Renal presentations: it include nephrolithiasis (**30-80%**), or nephrocalcinosis (**5-10%**) which is irreversible & may lead to renal failure.

3. Bone disease

- The common skeletal involvements are **subperiosteal resorption** especially of the **phalanges** and in severe cases cysts and tufting of terminal phalanges.
- Multiple pathological fractures may occur.

((It is said that the patient is losing his skeleton in his urine))

4. Gastrointestinal manifestations. Peptic ulcer disease and pancreatitis may occur in hyper-parathyroidism cases.

5. Emotional disturbances.

Neurologic and psychiatric problems may occur.

6. Hyperparathyroid crisis

- occurs with high serum calcium 16-20 mg per 100 ml.
- It presents by rapidly developing muscular weakness, nausea, vomiting, weight loss, fatigue and drowsiness.



Differential Diagnosis of hypercalcaemia:

Causes of hypercalcaemia, other than primary hyperparathyroidism, are:

1. Malignancy: characterized by recent onset of hypercalcaemia:

a. Group I (30%): Haematologic malignancies as multiple myeloma and lymphomas.

b. Group II (50%): Solid tumours with lytic bone metastases as cancers of breast, lung, kidney and pancreas.

c. Group III (20%): Solid tumours without bone metastases.

Hypercalcaemia is attributed to their secretion of a humoral factor that binds to PTH receptor in bone (parathormone-like substance).

2. Other causes: milk alkali syndrome, hyperthyroidism, Paget's disease of bone, vitamin D intoxication and sarcoidosis.



Investigations

1. Serum calcium level:

- The normal level is 8.5-10.5 mg/100 ml, values above **13 mg/100 ml** are strongly suspicious.
- Ca and P are simultaneously evaluated together with plasma proteins.

2. Parathormone immunoassay: Elevated plasma level of PTH does not establish diagnosis of hyperparathyroidism except with hypercalcaemia.

3. Serum chloride to phosphate ratio above 33 suggests hyperparathyroidism.

4. Increased excretion of calcium in urine.

5. Serum alkaline phosphatase is raised with skeletal lesions.

Localization of the tumor:

1. High resolution ultrasound has an accuracy of 76%.
2. CT scan has 50% accuracy as and is helpful in mediastinal sites.
3. Technetium labeled sestamibi scan has **high accuracy** in the localization of parathyroid adenoma with a sensitivity greater than 80%.
4. Newer modalities of scintigraphy in combination with single photon emission computed tomography (**SPECT**), SPECT with computed tomography (SPECT/CT).
5. In patients with **previous neck exploration, selective venous sampling** from certain points along the big veins, e.g. innominate and internal jugular veins may be helpful.



1- It is unusual that both(Renal) lesions occur simultaneously. The patients complain of **backache, haematuria, passage of renal calculi** of calcium phosphate or oxalate.

2- 75% of patients with primary hyperparathyroidism have calcium stones .

3-**Hypertension** due to **renal** impairment is responsible for 30% of deaths in patients with persistent hypertension after parathyroidectomy.

4- Hyperparathyroidism is a disease of bones, stones, abdominal groans and psychic moans.

5- Serum calcium may be normal in hyperparathyroidism in cases of hypo-albuminemia, pancreatitis, vitamin D and magnesium deficiency, and excess phosphate intake. Correction of these disorders may declare hypercalcaemia.

Combining ultrasound and sestamibi scan has a sensitivity of 95%. Sestamibi scan has replaced thallium technetium scan.



Treatment

- Mild Asymptomatic cases usually need no treatment. **Only follow up.**
- Surgical treatment: **(Parathyroidectomy)**

Indications: is

- 1-Patients with classic symptoms of primary HPT
- 2-Parathyroid Crisis.

Recent guidelines recommend

parathyroidectomy for those meeting one of the following criteria:

- (1) Age less than 50 years.
- (2) unable to participate in appropriate followup.
- (3) serum calcium level >1 mg/dl above normal range.
- (4) urine calcium >400 mg per 24 hours.
- (5) creatinine clearance <60 mL/min.
- (6) complications of primary HPT.

2. Multidetector computed tomographic angiography

Computed tomographic angiography (CTA) is done by injecting an iodine rich contrast material through an artery (not an artery) and so it is less invasive than catheter angiography.

3. Magnetic resonance angiography (MRA)

MRI image is generated from water hydrogen atoms, which are present in 70% of the blood in body. MRA can visualize blood vessels without the injection of any contrast.

4. Digital subtraction arteriography (DSA)

This technique allows visualization of the arterial tree without arterial cannulation to avoid its possible side effects. An x-ray picture is taken and the image is subtracted to the computer.

5. Direct arteriography

This invasive procedure is only performed if there is a need for interventional procedures. The different techniques of direct arteriography include:

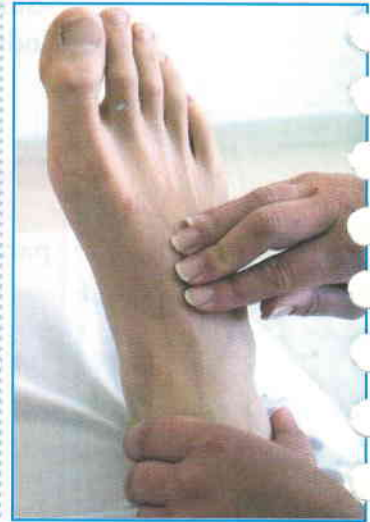
A. Direct femoral arteriography: direct puncture of the artery is done and injection of the contrast material will allow visualization of the whole arterial tree. This technique is being regularly nowadays in favor of the more informative angiography.

B. Trans-femoral aortography: A cannula is introduced into the femoral artery (the pulse is felt on this side), through it a guide wire is introduced. The cannula is removed and then a long catheter is introduced along the guide wire all it reaches the desired level and the contrast material is injected (Selinger technique). Trans-catheter arteriography is only performed if the whole distal aorta is excluded. Transcatheter arteriography by direct puncture of the aorta is no longer part of modern practice.

ARTERIAL DISORDERS

The effects of ischaemia depend upon:

- 1. The type of artery:** e.g., the subclavian artery thanks to the collateral circulation around the scapula. On the other hand, the popliteal artery has poor collateral circulation.
- 2. The rate of occlusion of the artery:** Acute ischaemia is much more serious than chronic ischaemia as there is not enough time for the collateral circulation to open.
- 3. The state of the collateral vessels:** Healthy collateral vessels can compensate to some extent the ill effects of ischaemia.
- 4. The general condition of the patient:** The presence of myocardial insufficiency or severe anaemia exacerbate the effects of ischaemia.



Vascular laboratory:

1. Doppler ultrasound:

Doppler ultrasound is noninvasive, painless, portable and inexpensive technique.

2. Multidetector computed tomographic angiography:

Computed tomography angiography (CTA) is done by injecting an iodine rich contrast material through an arm vein (not an artery) and so it is less invasive than catheter angiography.

3. Magnetic resonance angiography (MRA):

MRI image is generated from water hydrogen atoms, which are present in 70% of the human body. MRA can visualize blood vessels **without** the injection of any contrast.

4. Digital subtraction arteriography (DSA):

This technique allows visualization of the arterial tree **without** arterial cannulation to avoid its possible side effects. An x-ray picture is taken and the image is introduced to the computer.

5. Direct arteriography:

This **invasive** procedure is only performed if there is a need for endovascular procedures. The different techniques of direct arteriography include:

A. Direct femoral arteriography: direct puncture of the artery is done and injection of the contrast material will allow visualization of the whole arterial tree. This technique is losing popularity nowadays in favour of the more informative aortography

B. Trans-femoral aortography: A cannula is introduced into the femoral artery (the pulse is felt on this side), through it a guide wire is introduced. The cannula is removed and then a long catheter is introduced along the guide wire till it reaches the desired level and the contrast material is injected (**Seldinger technique**). Trans-axillary aortography is only performed if the whole distal aorta is occluded. Translumbar aortography by direct puncture of the aorta is no longer part of modern practice.



Before performing CTA, blood urea and creatinine should be estimated because the contrast material is nephrotoxic.

Arterial Embolism

Acute Limb Ischaemia

Acute ischaemia is a serious condition that occurs due to sudden interruption of the blood flow to a limb. If the treatment is delayed or unsatisfactory, many will lose a limb if not their life.

Aetiology:

1. Arterial embolism.
2. Aortic dissection.
3. Acute arterial thrombosis.
4. Extensive iliofemoral DVT.
5. Arterial trauma.
6. Compartment syndrome.
7. External compression (tight tourniquet).



- The usual source of emboli is a thrombus present either in the left side of the heart or in a proximal artery.
- An embolus in the arterial tree is usually arrested at the sites of bifurcation of arteries where sudden reduction in size of the arterial lumen occurs. The lower limbs are more commonly affected. The commonest sites of arrest of emboli in descending order of frequency are.



Definition

Embolus is originally a Greek word that means a plug or a stopper. Embolism is the passage of a matter from one part of the circulation to another through a vascular lumen.



Sources

- **Cardiac emboli (about 80-90% of cases):** These occur secondary to:
 - o **Atrial fibrillation:** is the **commonest** cause as it commonly results in stasis and the formation of a left atrial thrombus.
 - o **Myocardial infarction:** This leads to formation of a mural thrombosis.
 - o **Less common causes:** include left ventricular aneurysm, & endocarditis on top of rheumatic or congenital heart diseases.
 - **Non-cardiac emboli (about 20%):** Extensive atherosclerotic lesions or aneurysms of major arteries as the aortic arch or descending thoracic aorta.
- Sites of arterial embolism:
1. Common Femoral Artery bifurcation (40%).
 2. Aortic bifurcation (saddle embolus).
 3. Popliteal Artery bifurcation.
 4. Brachial Artery bifurcation.
 5. Common Carotid Artery bifurcation.

Acute Arterial Thrombosis



Aetiology

1. **Atherosclerotic obstruction:** Acute thrombotic occlusion on top of atherosclerotic narrowing is the **most common cause**.
2. **Pump failure:** A sudden reduction in cardiac output may produce acute limb ischaemia in patients with extensive atherosclerotic peripheral vascular disease.
3. **Hypercoagulable states:** can cause arterial occlusion, usually in small arteries.
4. **Febrile illness:** or gastroenteritis especially in children may lead to haemo-concentration with subsequent thrombosis.
5. **Acute graft thrombosis:** may occur early due to a technical factor or late due to progression of the original disease.
6. **Inadvertent intra-arterial injection:** which may occur in addicts causes intensive vasospasm followed by thrombosis.

Sequaele of acute arterial obstruction

1. **Propagation of the thrombus:** After circulatory arrest, widespread distal intravascular thrombosis occurs. The collateral circulation is also markedly impaired.
2. **Associated venous thrombosis:** This is due to a combination of sluggish flow and ischaemic injury to the intima of the involved veins.
3. **Compartment syndrome:** Ischaemic muscles get swollen, which in turn, exaggerates the effects of ischaemia. After revascularization, further oedema of the muscles occurs leading to more interruption of the circulation. Early fasciotomy of the muscle compartments can avoid this problem.

4. Systemic and metabolic sequelae: Muscle ischaemia leads to rhabdomyolysis with liberation of potassium, lactic acid and myoglobin. The sudden release of these metabolites into the systemic circulation will lead to “**reperfusion syndrome**” characterized by metabolic acidosis, hyperkalaemia, and myoglobinuria. Myoglobinuria and acidic urine lead to acute renal failure.



Clinical Picture of acute limb ischaemia (6P's)



Symptoms (3P's)

- 1. Pain:** is usually the first symptom. With embolism the pain is acute.
- 2. Paresthesia.**
- 3. Paralysis:** due to **ischaemic myopathy** more than nerve hypoperfusion. Loss of function of the intrinsic muscles of the foot occurs first. If ischaemia continues, patients develop calf tenderness due to swelling of the gastrocnemius and soleus muscles, which indicates muscle infarction.



Signs (3P's)

- 1. Pallor:** The colour of the skin is marble white with absent capillary refill.
- 2. Absent pulse:** is the **absolute sign** of acute ischaemia. Therefore, if peripheral pulses are felt arterial ischaemia can be excluded.
- 3. Poikilothermia (coldness).**

DIFFERENCES BETWEEN EMBOLIC & THROMBOTIC ACUTE ISCHAEMIA.		
	Embolism	Acute thrombosis
History	Arrhythmia or recent myocardial infarction	Claudication
Source of emboli	Usually present	Absent
Radial pulse	Usually irregular (AF)	Usually regular
Skin colour	White	Dusky
Limb nutrition	Normal	Picture of chronic ischaemia
Angiography	Sharp cut-off Minimal collaterals	Tapering stenosis Diffuse atherosclerosis Extensive collaterals

In acute ischemia, veins are collapsed and there is venous guttering. The leg may even appear cadaveric. Fixed mottling and fixed cyanosed or purple areas of skin that fail to blanch on pressure indicate that the capillaries have thrombosed and ruptured. Such limbs are unsalvageable.



The sensitivity of various tissues to ischaemia is variable. In a descending order as follows:

1. Sensory nerves responsible for light touch and proprioception are very sensitive followed by fibers responsible for pain, temperature and pressure.
2. Motor nerves leading to muscle weakness.
3. The skin. At first it is pale, then dusky blue due to capillary vasodilatation, lastly there is extravasation of blood due to capillary disintegration. At this stage, the skin is not viable.



Investigations (should be urgent)



Imaging

- 1. Duplex scan:** localizes and identifies embolism or thrombosis.
- 2. Arteriography:** may cause a delay of 2-3 hours. So, **it is not done in a threatened limb**. CT angiography or transfemoral arteriography can be performed:
 - **In embolism:** the proximal arteries are normal and there is abrupt occlusion (classic meniscus sign) and there is absence of collateral arteries.
 - **In acute thrombosis:** the proximal arteries are unhealthy and there is extensive collateral circulation. Angiography will show the distal run-off.

- 3. Echocardiography:** detects cardiac sources of embolism. The trans-oesophageal route is four times more informative than the trans-thoracic.



- Embolic acute ischemia usually indicates urgent surgery.
- Acute thrombosis may produce less severe ischemia because previous chronic ischemia opens collaterals.
- In evaluating viability of the limb:
 - a. Viable:** No sensory loss or muscle weakness, with audible arterial & venous Doppler signals.
 - b. Immediately threatened:** (Salvageable with immediate revascularization). Sensory loss is marked with rest pain, moderate muscle weakness, usually inaudible arterial Doppler signals, but audible venous Doppler signals.
 - c. Irreversible:** Profound sensory loss, profound muscle weakness or paralysis and inaudible arterial or venous Doppler signals. This stage is non-salvageable.



Labs

1. Intravascular hypovolaemia: leads to raised hemoglobin, blood urea nitrogen (BUN) and creatinine due to fluid sequestration in the limb.

2. Extensive muscle necrosis: leads to metabolic acidosis and raised creatine phosphokinase and WBCs.



Treatment

General measures:-

1. Control of arrhythmias or heart failure and cardiac monitoring.
2. Correction of dehydration.
3. control of pain.
5. **Oxygen administration.**
6. **Immediate heparinization** prevents proximal and distal thrombus formation. start with a loading dose of unfractionated heparin 80 IU/Kg followed by maintenance dose of **18 IU/ Kg/hour**. The dose is controlled by checking activated partial thromboplastin time (**APTT**) every 12 hours which should be maintained at **2.5-3 times** the baseline level.

Embolism:-

1. Urgent embolectomy using **Fogarty balloon catheter** is the standard method for removal of arterial emboli.
2. The operation should be done **as long as the limb is viable**. Because delay leads to obstruction of collaterals by propagation of thrombosis.
3. The operation can be done under local, general or epidural **anaesthesia**.
4. For embolism of the **femoral artery** embolectomy is done via a common femoral transverse arteriotomy.
5. For **aortic bifurcation** embolism, embolectomy is done via bilateral femoral arteriotomies.
6. For **brachial artery** embolism embolectomy is done via a brachial artery exposure.
7. **Arteriography** is done on table to confirm clearance of the arterial tree.
8. **Heparin** should be continued postoperatively until the cardiac condition is assessed and the need for further heparin therapy is determined.
9. **Fasciotomy:** Embolectomy may be followed by compartment syndrome. Therefore, it should be decompressed by full-length skin and fascial incisions from the knee to the ankle.
10. The **source** of arterial emboli should be corrected, if possible.

Acute Thrombosis:- (There is time to investigate)

1. Severe ischaemia: Urgent arteriography is performed to plan for **emergency revascularization** surgery.

2. Moderate ischaemia: Urgent arteriography is performed. There is time for the use of **thrombolytic therapy**. After dissolution of the thrombus **elective revascularization** surgery is done. Elective surgery carries a better prognosis than emergency surgery in these cases.

Arterial Injuries



Incidence

Arterial injuries are increasingly common nowadays:

1. The increased motor car accidents, war injuries & civil violence.
2. Iatrogenic injuries are now increasing with the introduction of invasive techniques, for the diagnosis and management of cardiovascular disorders.



Aetiology

- 1. Penetrating injury:** e.g. Stabs (Low velocity), Bullets (High velocity) passing through or near-by an artery “near miss”. Shotgun pellets may embolize in the arterial or venous systems.
- 2. Blunt injuries:** as in RTA or fall from heights. They injure arteries directly or through fractures.
- 3. Iatrogenic:** Following Arterial cannulation, Cardiac catheterization, Dialysis & Pelvic operations.
- 4. Intra-arterial drug injection.**

Pathological types

1. Arterial tear:

a. Complete tear (transection): the transected ends retract, constrict & thrombose → ischemia rather than hge.

b. Partial tear:

- The coats retract → widening of the gap → presents with hge.
- If bleeding remain within the tissues → pulsating hematomas → traumatic false aneurysm (pseudoaneurysm).
- If bleeding finds its way to the exterior (ext. wound, peritoneal or pleural cavities) → life threatening hge may occur.
- Less commonly concomittent injury to arteries & veins occur → A-V fistula.

2. Arterial contusion & thrombosis:

- Commonest type in closed injuries.
- Thrombosis occurs over the damaged area.
- Presents with ischemia.

3. Arterial spasm:

- Presents with ischemia.
- It should not be diagnosed except after artery exploration to exclude contusion & thrombosis.



Clinical Picture

Local examination is the basis for diagnosing arterial injuries.

• **Hard signs.** These are sure signs of arterial injury:

1. External arterial **bleeding**.
2. **Loss of distal pulses**.
3. Classic manifestations of **acute ischaemia (6Ps)**.
4. **Pulsating or expanding haematoma**.
5. A palpable **thrill** or an audible bruit.

• **Soft signs.** These are less specific (equivocal) signs:

1. Small or moderate sized haematoma that is **not pulsating & not expanding**.
2. **Proximity** of penetrating wound to a major vascular structure.
3. Adjacent **nerve injury**, producing neurological deficit.
4. **Diminished pulse** compared to the contralateral site.
5. History of **prehospital haemorrhage** that has stopped, or presentation with shock that cannot be explained by other injuries. In these cases, the patient could have bled from an arterial injury and the bleeding could have stopped before hospital admission.



Investigations

In patients with hard signs: diagnostic studies are unnecessary. Immediate surgical exploration is indicated as the delay in treatment is dangerous.

In patients with soft signs: urgent investigations are needed.

1. Plain X-ray: to detect foreign bodies or fractures.

2. Arteriography: is done only for patients with either an abnormal extremity pulse examination or a Doppler index less than 1.0.

3. CT angiography: visualizes the arterial injury and the adjacent surrounding structures.



Treatment

Immediate treatment

1. Control of external bleeding:

- Direct local compression of the site of injury is the most effective method at the scene of accident or in the emergency room.
- The blind placement of clamps into a bleeding wound should be avoided.
- Tourniquets should be avoided since they can occlude collateral inflow.
- Embedded knives or haematomas should not be disturbed.

2. Resuscitation: by blood and IV fluids follows the standard measures.

3. Heparinization: Systemic heparin is recommended only in patients with isolated vascular injury presenting with ischemia. Avoid in patients with multiple injuries especially those involving CNS, eyes & bone.

4. Prophylactic antibiotics: should be given early.

Definitive treatment

Time is the most crucial element that determines limb salvage certainly within **6 to 8** hours of injury.

- Arterial repair should be performed only by surgeons **experienced** in the techniques of vascular repair.

Methods of arterial repair include:

1. Clean-cut arterial injuries:

- a. **Longitudinal tears** are closed with vein patch to avoid lumen narrowing.
- b. In **transverse incision** $< \frac{1}{2}$ the circumference, the injury is repaired.
- c. If $> \frac{1}{2}$ the **circumference** is injured, complete division is performed then end to end repair.
- d. In **completely divided**, direct end to end anastomosis is done.

2. If the artery is contused or divided:

- o **Damaged edges** or segments are excised.
- o **Patch Angioplasty**: Reconstruction with an interposition graft (vein or less often syntnetic).

3. If the artery is found spastic:

- o Local application of **papaverine** is tried.
- o If not successful, **forcible dilatation** using Fogarty catheter or vessel dilator is performed.
- o If spasm still persists, **excision** of spastic segment with a saphenous vein graft is performed.

- **Local heparin** should be used routinely.
- Injured main veins are repaired **before the arteries**.
- Adequate **skin covering** is essential.
- **Fasciotomy** is done in:
 - o Late cases.
 - o Combined arterial and venous injuries.
 - o Muscle oedema.
 - o Development of swelling or paralysis after revascularization.
- In cases of displaced **fractures** pulses frequently return after reduction (within one hour). If pulses do not return within this Period of time, the artery should be explored.



One should never assume before exploration that a spasm is present & that it will be relieved with time. This serious error resulted in many limb losses. If vascular injury is found it should be repaired. Any associated fractures should be immobilized by metal fixation prior to the performance of a vascular anastomosis. For simple (closed) fractures internal fixation is permissible. For compound fractures, however, it carries the risk of introducing infection in the bone. In these cases, external metal fixation is done.



Drug addicts may wrongly inject drugs into arteries instead of veins. Anesthetists may also wrongly inject thiopentone into an artery during induction of anaesthesia. The most commonly punctured arteries are the brachial and the radial arteries. The drug flows into the distal small arteries leading to intense inflammatory reaction followed by thromb

Intra-Arterial Drug Injection



Clinical Picture

- Burning discomfort extending from the point of injection to the tips of the fingers occurs immediately after injection. This is rapidly followed by severe pain & blanching.
- Oedema develops rapidly and may be severe.
- The distal pulses are often normal.
- The hand is typically held in a claw like position. Sensory loss is usually present. The entire wrist and hand might be involved, but changes are most severe in the distal digits. The fingers are usually cool & deeply cyanotic.
- Digital gangrene & less commonly total extremity gangrene may develop.
- Arteriography should not be done as it will not add any more information to the clinical assessment.



Treatment

(should start immediately)

- Heparin at a dose of 10,000/1.U. intravenously, followed by a constant infusion to keep the partial thromboplastin time at 1.5-2 times. In cases of intra-arterial thiopentone injection the anaesthetist should not remove the needle from the artery before injecting the heparin bolus intraarterially. Heparin prevents thrombosis of small vessels.
- Dexamethazone 4mg I.V. every 6 hours limits oedema and tissue pressure elevation.
- Low molecular weight dextran (dextran 40) minimizes platelet aggregation.
- Strong analgesics.
- Extremity elevation to reduce oedema.
- Early passive and active exercises.

Chronic Limb Ischaemia

This is a slowly progressive arterial obstruction that gives enough time for collaterals to develop and, therefore, gangrene does not occur rapidly.



Aetiology

- Above the age of 45 years, atherosclerosis is the commonest cause.
- Below the age of 45 years
 - In diabetics presenile atherosclerosis is the main cause.
 - In non-diabetics
- In females Raynaud's disease in the upper limbs and arteritis in both the upper and lower limbs.
- In males Buerger's disease in the lower limbs and arteritis in both the upper and lower limbs.

Risk factors

- **Non-modifiable:** age, male gender, black race and positive family history.
- **Modifiable:**
 - Major: Hypertension, hypercholesterolaemia, smoking & DM.
 - Other: hypertriglyceridemia, obesity, sedentary or stressful lifestyle.



By far the most frequent cause is atherosclerosis.

Pathogenesis of atherosclerosis

- Subintimal deposition of low density lipoproteins (LDL) fatty streaks makes the earliest lesion.
- Endothelial dysfunction
- Proliferation of smooth muscle of media due to release of growth factors as platelet derived growth factor and fibroblast growth factor.
- Eventually, formation of a fibrous plaque leading to arterial narrowing.
- Complete arterial block may occur due to thrombosis on top of the atheroma or due to haemorrhage in the subintimal plaque.

Distribution

- Atheromas most commonly occur adjacent to arterial bifurcations, at the origin of major arterial branches and at sites where an artery passes beneath or through a fascial sling. These factors may cause disturbance of the laminar blood flow and cause abnormal eddy currents.
- The lesions have a special distribution. In descending order of frequency, they affect coronaries, cerebrals & carotids, arteries of lower limbs, the renal, superior mesenteric, coeliac arteries & last are arteries of upper limbs.
- Atherosclerosis that causes lower limb ischaemia affects one or more of the following three areas.
 - The aortic bifurcation (aortoiliac disease).
 - The superficial femoral artery (femoropopliteal disease).
 - The leg arteries (tibial disease).

Complications

- Chronic ischaemia due to gradual arterial occlusion.
- Acute ischaemia.
 - Ulceration of atheromatous plaques invites platelet aggregation which later detach and cause distal embolism.
 - Acute thrombosis on top of atheroma leads to complete arterial block.
 - Haemorrhage may occur under intima (another cause of arterial block).
 - Aneurysm formation.

Endothelial dysfunction

- Decreased nitric oxide which counteracts free oxygen radicals.
- Release of pro-coagulant factors.
- Release of inflammatory mediators.
- Release of angiotensin II



DD of chronic lower limb pain

1. Venous claudication

incompetent deep or perforating veins.

2. Neurogenic claudication

due to a herniated disc or spinal canal stenosis. The pain radiates from the gluteal region to the foot. The patient has to bend forwards or to lie flat on a hard matrix to relieve the pain.

3. Osteoarthritis

of the hip or knee joints. Fate of intermittent claudication. 70% remain stable, 20% deteriorate with slow progression to shorter walking distances, 10% develop critical limb ischaemia and 2% lose their limbs.



Clinical Picture



Symptoms

1. Intermittent claudications:

- **The earliest symptom:** Cramping pain occurs on walking & relieved by rest.
- **The claudication distance:** the distance which the patient can walk until pain occur.
- **The claudication time:** the time which the patient can walk until pain occur.
- **As the disease progress:** the claudication distance gradually shortens and the period of rest increases in length.
- **Site of pain depends on site of obstruction:** "Gluteal, Thigh & Calf pain in aortic bifurcation block, Calf pain in superficial femoral block. Foot pain in distal arteries block"
- **Mechanism:** Accumulation of metabolites due to muscle ischemia during walking. These metabolites are gradually washed away during rest.

Leriche Syndrome

A triad of:

1. claudication in one or both lower limbs.
2. Reduced sexual potency in male patients.
3. diminished or absent femoral pulses.



- Normally the dorsalis pedis artery is absent in 10% while the posterior tibial pulse is absent in 2% of individuals.
- Simple clinical examination can usually detect the site of arterial occlusion, e.g., in femoropopliteal occlusion the pain affects the calf muscles, trophic, colour and temperature changes are evident in the foot and leg. The femoral pulse is palpable while all distal pulses are not felt.

2. Rest pain: With further progress of the disease, the blood supply of the limb cannot even satisfy the requirements at rest:

- o **Ischaemic neuritis:** causes agonizing burning pain mainly in the foot.
- o Pain is more **severe at night** when the patient sleeps probably due to further diminution of the blood supply that is caused elevation of the foot. Another factor is that the warmth of the limb during sleep increases the metabolic demands while the blood supply cannot cope with these increased requirements. Warmth also induces cutaneous vasodilatation which further deprives the nerves of their blood supply. Many of these patients spend the night sitting in a chair in an attempt to relieve the pain.

4. Ulceration and gangrene: In severe ischaemia, superficial painful erosions may occur between the **toes**, on the dorsum of the foot, or around the malleoli. The end stage of ischaemia is dry gangrene.

5. Impotence: occurs in **aortic bifurcation** block due to diminished blood flow in the internal iliac arteries (Leriche Syndrome).

6. Atherosclerosis symptoms: angina pectoris, cerebral stroke or transient ischaemic attacks and postprandial abdominal pain (**intestinal ischaemia**).



Signs

General examination

- 1. Pulse:** All accessible arteries are palpated to assess the presence of the pulse, any arrhythmias and the condition of the arterial wall. A stethoscope over a stenosed artery may reveal a murmur.
- 2. Blood pressure:** should be measured.

Local examination

1. Trophic changes:

- o **Loss of skin appendages:** (hairs and sebaceous & sweat glands): skin becomes dry. The nails become brittle, deformed & lose their luster.
- o **Loss of subcutaneous fat:** skin becomes thin & toes become tapered.
- o **The muscles:** become wasted and the bones osteoporotic.
- o **Ischaemic ulcers:** may develop at the tips of the toes.

2. Temperature changes: Some degree of coldness will be detected in the ischaemic limb up to a level below but near that of obstruction.

3. Colour changes: Pallor, cyanosis and sometimes, redness (rubor) may occur.

- **Pallor:** is due to decreased blood flow into the skin.
- **Cyanosis:** and redness are due to stagnation of blood in the markedly dilated capillaries under the effect of accumulated vasodilator metabolites.
- **Rubor:** The colour of blood is at first red but it later becomes blue due to extraction of oxygen by the tissues.

4. Absence of pulses: The pulses distal to the site of occlusion will be absent or markedly weak. A murmur may be heard over the site of the stenotic arterial segment.

• Special tests:

o Test for the capillary circulation: If one presses over the tip of the toe, it becomes pale. Once the pressure is released, the colour returns within two seconds. In an ischaemic limb the return of colour is slow and is called sluggish capillary circulation.

o Buerger's angle: The patient lies supine and the limb is gradually elevated. The angle at which blanching of the toes occurs is called Buerger's angle. Normally, blanching does not occur even when the limb is raised up to 90°. The smaller the angle at which blanching occurs, the more severe the ischaemia is.

o Harvey's venous refilling time: With the patient supine, the limb is elevated to right angle until all veins empty. It is then brought down to the horizontal position. Normally the veins fill in 10-15 seconds, in chronic ischaemia venous refilling is delayed to above 30 seconds.



Investigations



Labs

1. Blood sugar.
2. Blood picture to detect anaemia or polycythaemia.
3. Serum lipids.
4. Serum creatinine.
5. ECG and fundus examination.



Imaging

1. Doppler flow study: Values:

- Stenosed or occluded segments can be detected.
- Detecting collateral refilling of post-stenosed or post-occluded segments.
- Measurement of the Ankle/Brachial (AB) index. By using the ultrasound probe the blood pressure at the ankle and brachial arteries can be measured.
- Segmental pressures of the different arterial segments can be measured. normal arteria flow gives a triphasic wave & the collateral flow gives a biphasic wave.

2. Duplex scanning: This examination can visualize the arteries and detect areas of stenosis or block. It combines the benefits of Doppler plus ultrasound images of the vessels.

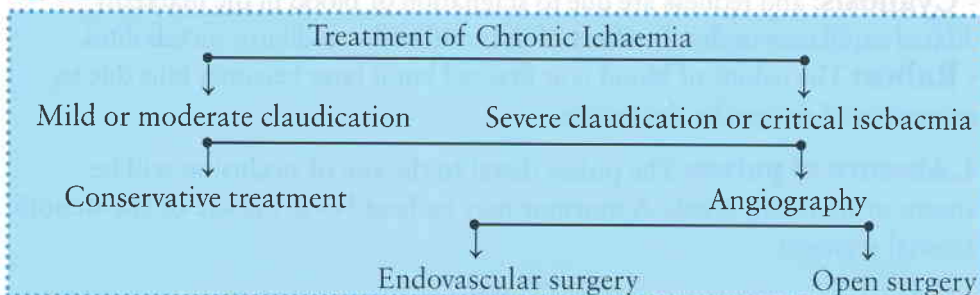
3. CT angiography (CTA).

4. Magnetic resonance angiography.

5. Direct arteriography: is performed when endovascular surgery is intended.



Treatment



Clinical staging of chronic limb ischaemia

- **Stage I:** Asymptomatic
- **Stage II:** Intermittent claudication
- **Stage III:** Rest pain
- **Stage IV:** Ulceration or gangrene

Critical limb ischaemia

(CLI) is diagnosed as persistent recurring ischaemic rest pain requiring opiate analgesia for at least 2 weeks, ulceration or gangrene of the foot or toes, and ankle systolic pressure less than 50 mm Hg or toe systolic pressure less than 30 mmHg.

AB Index

1-1.2	Normal
< 0.9	Ischaemia.
< 0.7	Severe Ischaemia.
< 0.3	Impending gangrene.

Points essential if a bypass procedure is planned:

- i. Site of arterial block.
- ii. State of proximal arteries.
- iii. Visualization of the artery distal to the block (run off).



Conservative

1. **Mild exercises** short of causing pain are useful as they help open the collateral circulation.
2. Complete abstinence from **smoking**.
3. Correction of **anaemia** as this helps tissue oxygenation.
4. **Control** of diabetes mellitus, hypertension and hyperlipidaemia.



Medical

1. **Aspirin (150 mg/day)** reduces platelet adhesiveness.
2. **Statins** to control hyperlipidemia.
3. **Calcium Channel Blockers**.
4. **Cilostazol** reduces platelet aggregation. It also raises the level of HDL cholesterol and lowers triglycerides.



Surgical

Indication for surgery:

1. **Severe claudications** that interfere with the pt's work & lifestyle e.g. farmer not clerk.
2. **Critical ischemia** (ischemic ulcer, rest pain or minor gangrene) to save the limb (Limb salvage surgery).

A- Endovascular surgery:

Indications: Localized obstruction in large & medium sized arteries.

Methods:

1. **Percutaneous transluminal angioplasty (PTA):** A special balloon catheter is introduced percutaneously until the balloon reaches the stenosed or occluded segment. It is then inflated & kept for about 1 min to dilate the stenosed segment.

Indications: < 2 cm significant stenotic segments in otherwise relatively normal arteries.

Success rates: about 95%.

Complications: Hematoma, AV fistula, Restenosis.

2. **Application of stent:** After balloon angioplasty, a stent can be inserted to prevent elastic recoil of the arterial wall & keep the lumen patent.

B- Open Surgery:

1. Thromboendarterectomy:

o Removal of the thrombus, intima & inner media leaving a patent lumen that becomes rapidly endothelialized

o Indication: Large artery with localized obstruction e.g. a localized narrowing in an iliac artery.

2. Bypass surgery (more frequently performed):

Idea: is to bypass the obstruction by inserting a graft from the healthy artery above to another healthy artery below the obstruction.

Indication: Big & medium sized artery with extensive or multiple lesions involving a long segment.

- For Aorto- iliac disease → Aorto-bifemoral bypass using a synthetic graft [Dacron or polytetrafluoroethylene (PTFE)].
- For Superficial femoral artery block → Femoro-popliteal bypass.

- The saphenous vein is the best graft for this operation.
- It has valves that allow blood to flow from below upwards only. This problem is overcome by:
- Reversed long Saphenous vein graft: to prevent obstruction by its valves.
- In situ long saphenous vein graft: valves are destroyed by Valvulotome & the vein tributaries are ligated to prevent A.V. fistula.

What to do for pts who don't have good distal run-off? (little to be done)

1. Lumbar sympathectomy doesn't improve bl. supply of the ms. So, it's mainly indicated when there are cutaneous ulcers or gangrene of the foot. It's also useful for rest pain.
2. IV or Intra-arterial PGE.
3. Amputation for gangrene at a proper level or wait for separation.

Indications for amputation:

1. Spreading or massive gangrene.
2. Spread infection.
3. Severe uncontrollable pain making the pt request amputation.

Level of amputation: Depends on the blood supply sufficient for healing of the wound:

- In atherosclerosis (involve femoral artery) above knee amputation (the stump will be supplied by the profunda femoris).
- If popliteal pulse is felt below knee amputation.

Thrombo-Angiitis Obliterans (Buerger's Disease)



- The disease has patchy distribution & episodic course.
- It affects vessels distal to the popliteal artery.
- The neurovascular bundle shows panvasculitis & neuritis.
- The artery is obstructed by a thrombus which becomes organized.
- Affection of the nerves by the inflammatory process and the early block of arteries explain the severe pain that is present in these cases.



Pathophysiology

1. The disease has **patchy** distribution and an **episodic course**.
2. It affects the vessels **distal** to the popliteal artery.
3. The **neurovascular** bundle shows **panvasculitis and neuritis**.
4. The artery is obstructed by a **thrombus** which becomes organized.
5. Affection of the **nerves** by the inflammatory process and the **early block** of arteries explain the **severe pain** that is present in these cases.



Clinical Picture

1. **Chronic ischaemia**: the disease is suspected when a **young male** who is heavy **smoker** complains of chronic ischaemia.
2. **Migrating thrombophlebitis**: may precede the disease, which appears as a **small, tender cord** along the course of a vein.
3. **Raynaud's phenomenon**: may be superimposed.
4. **Interdigital fungus infection**: is a common association.

Differences Between Atherosclerosis & Buerger's Disease.

	Atherosclerosis	Buerger's disease
Age	Elderly	20-40 years
Sex	Commoner in males	Exclusively in males
Aetiology	Main risk factors are • Hypertension • High cholesterol • DM	Excessive smoking
Level of lesion	Aorta-iliac, femoro-popliteal or distal	Distal vessels with patchy distribution
Pathology	Mainly intimal (atheroma)	Inflamed neurovascular bundle & thrombi that block lumen.
Migrating thrombophlebitis	Absent	Usually present
Rest pain	May be present but late	Marked early feature



Investigations

1. **Duplex scanning**: It reveals distal arterial occlusive disease with **corkscrew collaterals**.
2. **Digital subtraction angiography**: will reveal involvement of **small and medium sized arteries** as the digital, planter and proneal ones.



Treatment

1. **Smoking cessation**: to avoid disease progress.
2. **Sympathectomy**: provides short term pain relief and promotes healing.
3. **Amputation**: of one or more digits or toes is indicated for persisting pain or gangrene and can be performed **adjacent** to the **line of demarcation** with satisfactory primary healing.

Vasospastic Disorders

Raynaud's Disease



Aetiology

(Not exactly known. Certain factors are suggested)

1. **Abnormal sensitivity** of the small arteries and arterioles of the hands, and less commonly the feet, to **cold**.
2. **Increased sympathetic tone**.



Clinical Picture

For the **diagnosis** of Raynaud's disease certain **criteria** should be fulfilled.

1. **Bilateral** and **Symmetrical**.
2. Common in **young females**, in both hands.
3. The attacks are **precipitated** by **coldness** and are relieved by warmth.
4. The attack consists of 3 consecutive phases
 1. **Pallor**: due to spasm of the digital arterioles.
 2. **Cyanosis**: due to dilatation of the capillaries which are filled with slowly flowing deoxygenated blood.
 3. **Redness**: As the attack passes off, arterioles dilate & oxygenated blood passes into the dilated capillaries: The attack is accompanied by pain and is called Raynaud's phenomenon.
5. **Radial & Ulnar** pulses are **preserved**.
6. **No major gangrene**: Only minute patches of ulceration or gangrene.

Grades of Raynaud's disease:

- 1st → **Only Raynaud's** phenomenon.
- 2nd → **Mild trophic** changes in the tips of fingers and nails.
- 3rd → **Gangrene** of the tips of fingers.



Treatment

1. **In early stages**: conservative measures are tried:
 1. **Avoid** cold weather and to wear woolen gloves in winter time.
 2. Vasodilator **drugs**.
 3. Calcium channel **blockers**.
2. **In severe cases**: cervico-dorsal **sympathectomy** is performed. Its immediate results are good but the symptoms usually recur after sometime. Recurrence, at least, is not severe.

Raynaud's Phenomenon

Colour changes, similar to those of Raynaud's disease, may accompany a number of conditions:

- Thoracic outlet syndrome.
- Certain occupations as typists, pianists and labourers who use vibrating tools.
- Collagen diseases as rheumatoid arthritis, scleroderma, systemic lupus erythematosus and dermatomyositis.
- Vascular disorders as atherosclerosis and Buerger's disease.
- Drugs as chronic administration of ergot-containing drugs for migraine, and ergot poisoning.
- Atrophic disorders of limbs, e.g., after poliomyelitis.

Treatment

- should be directed to the original problem.
- Vasodilators and 8-blockers are prescribed.

Diabetic Foot Infection And Ulceration

25times

diabetics are susceptible to foot problems more than normal persons.

70%

of limb amputations are performed on diabetics.



Aetiology

1. Peripheral neuropathy: is present in 25-40% of patients.

This neuropathy may be:

a. Sensory neuropathy: the patients are unaware of injurious insults.

b. Motor neuropathy: of the intrinsic muscles of the foot leading to clawing of the toes and subluxation of the metatarsophalangeal joints, which puts undue pressure on the metatarsal heads. A callosity forms over the pressure areas and underlying infection occurs. It results in a neuropathic ulcer.

c. Autonomic neuropathy: Sympathetic denervation leads to peripheral vasodilatation and A.V shunting. Sweating is suppressed leading to cracking and fissuring of the skin.

2. Ischaemia: premature atherosclerosis of major vessels & micro-angiopathy diminish the blood supply and nutrition of the tissues.

3. Hyperglycaemia and compromise of the immune system:

Both humoral and cellular immunity are inhibited in diabetic patients.



Clinical features

1. Neuropathic ulcers: painless with punched out edges and with no signs of healing usually on the planter surface of the metatarsal head. Examination reveals peripheral neuropathy.

2. Ischaemic ulcers: usually in the distal parts of the toes. Symptoms and signs of chronic ischaemia are present.

3. Infection: usually follows a minor trauma e.g. a pin prick, careless cutting of a nail, scraping of a callosity or interdigital infection. Infection spreads rapidly leading to cellulitis, necrosis of tendons and muscles and osteomyelitis. Multiple pockets of pus are present. In severe cases localized or massive gangrene of the toes or foot may occur.



Prevention

1. Adequate control of diabetes.
2. Careful trimming of toe nails.
3. Avoidance of tight foot wear.
4. Early treatment of tenia pedis infection.
5. Daily inspection of the feet to look for wounds & interdigital infections.
6. Avoidance of walking barefooted.
7. Daily feet care by washing, drying, powdering.



Investigations

1. Blood **sugar**.
2. **Plain x-ray** may show osteomyelitis, pathological fracture, Charcot's joint or gas in soft tissues.
3. Swabs for **culture** and sensitivity should be taken from any discharge.
4. **Duplex** ultrasound.
5. **CT angiography** if there is clinical evidence of ischaemia.
6. **MRI** to assess the condition of the soft tissues.



Treatment

1. Hospitalization.
2. **Rest** in bed and elevation of the foot.
3. Proper **control** of diabetes, better by crystalline insulin every 8 hours.
4. Broad spectrum antibiotics are started immediately until the results of culture are available.
5. **Drainage** of infection and debridement.
 - a. Under general anaesthesia:
 - Drain all pockets of pus.
 - All sloughs should be excised.
 - A gangrenous toe needs to be amputated leaving the wound open.
 - b. In cases with widespread infection, some form of a **major amputation** is necessary to save the patient's life.
 - c. Repeated **dressings** and debridement with drainage of pockets of pus.
 - d. When the wound is completely free of infection, a large raw area is considered for **plastic skin coverage** in order to shorten the recovery time.
6. Patients with clinical occlusion of a major artery, angiography is done to assess the feasibility of **vascular reconstruction**.



Diabetic foot is a serious problem which should never be treated lightly.

In many patients, anaerobic organisms are involved and should be covered.



Gangrene occurs at a younger age in patients who have diabetes, Buerger's disease or other types of arteritis.

• The five cardinal signs of gangrene are remembered by "Press and See How Colour Fades".

1. Loss of **P**ulsation and sluggish capillary circulation.
2. Change of skin **C**olour into blue and later black. The colour does not change by local pressure. This is referred to as "fixed colour changes".
3. Loss of **H**eat (coldness).
4. Loss of **S**ensation.
5. Loss of **F**unction.

Consequences

Gangrene is a serious condition. If untreated, death may occur from progressive spread with severe toxæmia.

Gangrene

It is **macroscopic** death of tissues that is generally caused by loss of blood supply & is usually associated with bacterial invasion & putrefaction.

+ Aetiology

• **Ischaemic:**

- o **Thrombosis:** e.g., on top of atherosclerosis (senile gangrene).
- o **Embolism.**
- o **Vasospastic diseases:** e.g. Raynaud's disease and ergotism.

• **Neuropathic.** Diabetes mellitus, syringomyelia and leprosy.

• **Traumatic:**

- o **Direct trauma:** due to crushing or pressure (bed-sores).
- o **Indirect trauma:** due to injury of the main vessels.

• **Physicochemical:** Burns, frost-bite and trench foot.

• **Infective:**

- o **Specific infection:** Clostridial gas gangrene.
- o **Nonspecific infections:** as necrotizing fasciitis and carbuncle.

• **Venous gangrene.**

Clinical types

• **Dry gangrene:** occurs as a result of slowly progressive vascular disease, as in atherosclerosis (senile gangrene). The gradual slowing of blood flow permits evaporation from the affected surface and results in desiccation and mummification of tissues which become dry, wrinkled, shrunken and black.

• **Aseptic moist gangrene:** occurs when the gangrenous process is associated with water logging of the tissues from **sudden occlusion** of the main artery by a **ligature** or embolus, coincident **venous occlusion** as in **traumatic gangrene**. The part remains of the same size & consistency but become discoloured, being white at first and later purple or greenish-black.

• **Septic moist gangrene:** due to **infection of sterile gangrene** (2^{ry} infective gangrene) or to infection of tissues with **virulent organisms** which bring about death of the infected tissues (1^{ry} infective gangrene). It becomes swollen, oedematous, and inflamed. Skin appears moist and blotchy & the epidermis is raised from the surface by bullae filled with serum. The part emits a **very offensive odour**, and may **crepitate** due to gas formation from rapid decomposition. There is marked inflammation in the living tissues above it.

Constitutional signs are present & death occurs from septicaemia.

Clinical Picture

- It is preceded by signs and symptoms of progressive ischaemia.
- Gangrene starts as an area of painful redness, in the center of which a slough forms and becomes dry and black. The slough may separate leaving an ulcer.
- The **great toe** is the commonest site of the disease which spreads to the foot or neighbouring toes or attacks the other toes independently.
- **Pain is marked** and causes insomnia and exhaustion.

Aneurysms

An aneurysm is a **sac** that contains blood and **communicates** with the lumen of an artery.

Practically an aneurysm is a permanent **localized dilatation** of an artery, having at least 1.5 times the normal diameter of that given segment.

Aneurysms can be classified according to:

- Aetiology: Pathological, traumatic and congenital.
- Structure: True or false.

True aneurysm: the wall is formed of the 3 layers of the dilated artery.

False aneurysm: is actually a haematoma communicating with lumen of an artery through a partial tear in its wall. Thus, the wall of the false aneurysm is formed by the fibrous wall of the haematoma.

- Shape: Fusiform, saccular or dissecting.

Aetiology

Aneurysms are caused by weakness of the arterial wall. Atherosclerosis is the commonest cause. Hypertension, if present, helps increasing its size.

- Pathological. Any disease which weakens the arterial wall may lead to aneurysmal dilatation.
 - o The commonest cause is atherosclerosis leading to aneurysmal formation at various sites. Common examples are the abdominal aorta and the popliteal artery.
 - o Less common causes are cystic medial necrosis, septic emboli of subacute bacterial endocarditis and collagen diseases as Behcet's disease, Marfan syndrome and Ehler's Danlos syndrome. Previously syphilis was a common cause of thoracoabdominal aneurysms.
- Traumatic
 - o Blunt trauma to an artery may weaken part of its wall. Later this weak area progressively yields leading to aneurysmal dilatation.
 - o A penetrating injury to an artery may cause a small hole in the wall leading to the formation of a haematoma next to the artery. Later on, this clot is surrounded by a false capsule of organized fibrous tissue and the result will be a false aneurysm.
- Congenital. These may occur in the circle of Willis (Berry aneurysms), where they may cause subarachnoid haemorrhage. Other sites include the splenic, renal or coeliac vessels.

Complications

- Rupture is the most serious complication. It can produce fatal haemorrhage.
- Distal ischaemia.
 - o Thrombosis and occlusion of the aneurysm.
 - o Detachment of fragments of this thrombus cause distal embolization. A common example is the occurrence of gangrene in one of the digits due to distal emboli from subclavian aneurysm which develops in patients with thoracic outlet syndrome.
- Infection may lead to rupture and secondary haemorrhage.
- Compression on adjacent structures. As the aneurysm gets bigger, it may compress the adjacent structures. Compression of an adjacent vein may cause obstruction or thrombosis. Compression of a nerve may cause motor or sensory affection. The adjacent bone may also be eroded.



Clinical Picture

- Silent aneurysms. Some aneurysms, e.g., of the abdominal aorta are commonly silent.

They are accidentally discovered at clinical or ultrasound examination that is done for another reason.

- Swelling.
- Symptoms due to compression on adjacent structures.
- Complications.
- Local signs of an aneurysm
 - A swelling that lies along the line of an artery and can be moved across the line of the artery but not along it.
 - The swelling exhibits expansile pulsations (expansile means expansion in all directions. This is the most important sign.
 - Proximal pressure on the main artery results in diminution or disappearance of the pulsations.
 - Distal compression on the main artery causes the aneurysm to increase in size and to become tenser.
 - A systolic thrill may be felt and a bruit may be heard.



Differential Diagnosis

- A swelling overlying an artery may elicit transmitted arterial pulsations. Pressure on the proximal artery does not change the size of the swelling. If the swelling can be moved away from the artery, the pulsations disappear.
- A very vascular tumour as an osteosarcoma or metastases may pulsate.
- An abscess.
- A serpentine artery as is sometimes the case with the common carotid.
- An arterio-venous fistula.



Investigations

1. Duplex scanning is very useful.
2. CT angiography is very accurate in the diagnosis of aneurysms.
3. Magnetic resonance angiography.



Treatment

Aneurysms are liable to rupture. Therefore, most aneurysms require treatment.

- The standard line of treatment is excision and graft.
 - Exclusion graft. Insertion of a graft inside the sac of the aneurysm without removal of the sac, this is commonly done for aortic aneurysms.
 - Excision with arterial ligation can be done for aneurysms of small arteries as the radial and ulnar arteries.
 - Endovascular treatment with stent insertion.

Abdominal Aortic Aneurysm (AAA)

This is the most frequent type of aneurysms. It affects the aorta below the origin of the renal arteries in 95% of cases. It may extend to affect the iliacs. Rarely does it extend upwards to involve variable distance of the suprarenal abdominal aorta or the thoracic aorta in which case it is called a thoraco-abdominal aortic aneurysm.



Aetiology

Atherosclerosis is the commonest cause and is responsible for 95% of AAAs.



Clinical Picture

- **Asymptomatic aneurysms:** In 75% of patients the AAA is discovered accidentally during a routine abdominal examination (as a pulsatile epigastric mass) or during ultrasonography or CT scan performed for some other reason.

- **Pain:** is the commonest symptom: An A.A.A. gradually enlarges and impinges on surrounding structures causing vague abdominal pain. Back and flank pain results from vertebral compression. Large aneurysms can even erode the spine and cause severe back pain in the absence of rupture. These patients may be wrongly diagnosed as having lumbar disc prolapse.

- **Symptoms of rupture:** The classic triad of AAA rupture is sudden severe pain, a pulsatile abdominal mass and shock. However sometimes one or more of the components of the triad are absent in a patient with rupture.

- **Acute upper abdominal pain:** The abrupt onset of severe pain in the back, flank or abdomen is characteristic of aneurysm rupture or acute expansion.

- **Pulsatile abdominal mass:** which is usually tender. It may be masked by obesity or abdominal distension.

- **Shock:** is present in most cases at the time of presentation. In other cases, rupture is so effectively contained within the retroperitoneum that hypotension may be absent at the time of initial examination. If the patient is left untreated shock may develop at any time.



Investigations

- **Ultrasonography:** If an AAA is clinically suspected, ultrasonography is the screening test of choice to document or to rule out the presence of an aneurysm. It is rapid, inexpensive, non-invasive and accurate.

- **C.T scan:** If repair of an AAA is decided, CT scan provides data that are important for surgery especially if endovascular repair is considered. In this respect, spiral CT is superior to the conventional axial CT as the former can display the information in multiple planes and allow three-dimensional reconstruction of the aneurysm sac.

- **MRI:** is a good alternative to CT scan but is costly.

- **Transfemoral aortography:** is of value if the patient has been suspected to have associated occlusive disease in the iliac, renal, or mesenteric arteries to plan the simultaneous repair of the occlusive disease.

Natural history of AAA

Rupture is the most frequent and most serious complication of AAA. The risk of rupture is mainly related to the size of the aneurysm. The 5-year rupture rates for untreated AAA of diameters 5cm or more is high, even if asymptomatic.



Treatment

Plan of management

- Immediate surgery for patients with the diagnosis of rupture. These should be taken to the operating room immediately for aneurysm repair. No time should be lost in a surgical ward or in an ICU.
- Urgent surgery for patients with symptoms of acute expansion (severe pain of acute onset but with no leak on C.T. scan).
- Elective surgery for
 - o Symptomatic aneurysms regardless of the size.
 - o Asymptomatic aneurysms 5cm or more in diameter.
- Regular follow up is indicated for patients with asymptomatic aneurysms less than 5cm in diameter. Ultrasound is done every 6 months. Aneurysm repair is required if serial studies show an enlarging aneurysm.

Type of surgery for aneurysm repair

- The standard is conventional open surgery by opening the aneurysm and excluding it by implanting a synthetic graft.
- Endovascular repair of AAA. This entails insertion of an endoluminal stented graft through bilateral femoral arteriotomies. This technique is particularly indicated for
 - o High risk patients who cannot tolerate anaesthesia and open surgery.
 - o High-risk local abdominal factors, e.g., previous major surgery.

Arteriovenous Fistula (A-V fistula)

This is an abnormal communication between an artery and a vein. It may be congenital or acquired.

Acquired arteriovenous fistula

This is usually secondary to a penetrating injury by a knife or a bullet which induces a communication between an artery and a neighbouring vein. A common site is the femoral triangle, as during angiographic procedures the cannula may injure the vessels leading to A-V fistula. In patients with chronic renal failure who need haemodialysis, an A-V fistula is intentionally made by the surgeon, usually between the cephalic vein and the radial artery.

This arterializes the superficial veins of the limb, i.e. they become distended with fastflowing arterial blood which makes them convenient for puncture and drawing out blood for haemodialysis.



Clinical Picture

- **Local signs:** There is a small swelling along the course of the vessels. The striking feature is that the swelling has a continuous thrill over it and auscultation reveals a continuous machinery murmur over the swelling and propagated along the vein.

- **Distal signs:** The veins become dilated, tortuous, thick walled and pulsating. Oedema, and congestion may appear.
- **Systemic signs:** If the fistula is of a moderate or large size, there will be marked reduction in the peripheral resistance and an increase in the venous return. These will lead to tachycardia, increased cardiac output with high systolic and low diastolic blood pressure (water hammer pulse). Local pressure at the site of the fistula causes marked slowing of the pulse (Branham's sign). A high output large fistula can lead to cardiac failure.



Investigations

- **Doppler:** can localize the site of the fistula.
- **Arteriography:** will show both the artery and vein at the same time due to rapid venous filling. It may also help localize the site of the fistula.



Treatment

The ideal treatment is by excision of the sac and restoration of continuity of both artery and vein.

Extracranial Cerebrovascular Disease

Cerebrovascular disease is the third leading cause of death and is a major source of disability among elderly people. Thromboembolic disease accounts for about 75% of stroke; of these, carotid artery occlusive disease is the most important cause.



Pathophysiology

- Embolization is the commonest cause of transient ischaemic attacks (TIAs) and ischaemic stroke.
- The embolus may arise from the heart, but more commonly it arises from an ulcerating atherosclerotic plaque at the carotid bifurcation. Ulceration and irregularity of the plaque stimulate platelet aggregation.
- If the platelet aggregates embolize to an important vessel in the brain, symptoms occur.
 - o If the platelet aggregates break up quickly the symptoms are transient and the patient experiences a TIA.
 - o If the embolic fragment persists, it can lead to focal infarction and the patient experiences a stroke.



Clinical Picture

Patients can be classified into three categories

- **Asymptomatic patients.** The atherosclerotic lesion in the carotid artery is discovered accidentally on a screening carotid duplex prior to major surgical procedures, e.g., coronary artery bypasses.
- **Patients with transient ischaemic attacks (TIAs).** Transient ischaemic attacks are defined as temporary focal neurologic or visual deficits lasting less than 24 hours and end with complete recovery. TIAs are of sudden onset and typically resolve within one hour.
 - o Carotid artery TIAs are caused by deficits in areas supplied by the anterior and middle cerebral arteries. Manifestations include one or more of the following

- Motor dysfunction as weakness or paralysis of one or both limbs contralateral to the affected hemisphere.
- Sensory alterations as numbness, loss of sensation or parasthesia in one or both contralateral limbs or in the opposite side of the face.
- Receptive or motor aphasia if the dominant hemisphere is affected (the left side in 95% of patients).
- Amaurosis fugax is ipsilateral transient loss of vision described by the patients as a black curtain coming across the eye.
 - Vertebrobasilar TIAs. Symptoms include vertigo, ataxia, dizziness, bilateral parasthesias and visual hallucinations.

• **Patients with stroke.** The patient has residual neurologic deficits that could be minimal or profound depending on the extent of brain damage. A stroke may be fatal.



Investigations

- **Duplex scan** is the first-line investigation to study the extracranial cerebrovascular arteries in a symptomatic patient. If done by a skilled operator it can give accurate information on the presence and the degree of stenosis.
- **CT angiography.**
- **CT scan of the brain** is done before operation to define the presence of pre-existing cerebral damage and to exclude other brain pathology.



Treatment



Surgical

The operation is carotid endarterectomy. The atherosclerotic plaque at the carotid bifurcation is removed surgically. By removing the plaque, the risk of a future stroke is markedly diminished.

Indications of carotid endarterectomy. Internal carotid artery stenosis of 70% or greater in a patient with

- Carotid artery TIAs.
- Stroke leaving minimal neurologic deficit.
- Asymptomatic lesion.

Medical treatment

Indications

- Less than 50% stenosis even if symptomatic.
- Patients during the acute phase of a stroke. Revascularization is contraindicated as it leads to haemorrhage in the infarcted brain area.
- Patients suffering from stroke with poor recovery.



Medical

- Antiplatelet drugs such as aspirin or clopidogrel. They inhibit platelet aggregation.
 - Stopping smoking & control of diabetes, hypertension and hyperlipidaemia.
 - Full anticoagulation in patients with cardiac embolic disease.
- Either medical or surgical treatment is acceptable option for symptomatic patients with 50-69% stenosis.

Subclavian Steal Syndrome

In this syndrome, cerebral ischaemia is caused by the reversal of flow in the ipsilateral vertebral artery distal to a proximal lesion in the first part of the subclavian artery.



Clinical Picture

Vertebrobasilar insufficiency occurs on exercise of the upper limb. This is due to increased demand for blood, which the extremity steals from the cerebral circulation through the ipsilateral vertebral artery.



Investigations

- Duplex scanning.
- CT angiography.



Treatment

Surgery: Options include

- Carotid-subclavian artery bypass.
- Percutaneous transluminal balloon angioplasty (PTA) of subclavian stenosis.

Endovascular Surgery

Endovascular surgery is the management of vascular diseases percutaneously through a puncture to deal with a lesion in a remote site.

Balloon angioplasty

1. Percutaneous transluminal angioplasty (PTA)

Indications

These are the same as those for conventional vascular surgery.

- Critical ischaemia as denoted by rest pain and the presence of ischaemic ulcers or minor gangrene.
- Severe incapacitating claudication that interferes patient's work & life style.

Ideal lesion

- At least 5mm beyond origin of an artery.
- Stenosis < 5cm length.
- Best in iliac artery.

The technique is also very useful as an adjunctive procedure with surgical revascularization.

2. Arterial stent

Aim: A stent may be inserted in the same session after PTA to prevent its drawbacks (dissection and elastic recoil) in order to maintain patency of the artery.

Principle

- A metal vascular stent is guided over a guide-wire, endoluminally, and is directed to the arterial segment that has been dilated.
- The stent is carried over an expanding device that is kept closed until the stent reaches the desired site. The device is then opened to expand the stent so that it will fit in the artery.

Inferior vena cava filter

Indications

- DVT or documented pulmonary embolism in a patient who has a contraindication to anticoagulation, e.g., previous intracerebral hemorrhage or history, bleeding peptic ulcer.
- Recurrent pulmonary embolism despite adequate anticoagulation.
- Complications of anticoagulation that forced therapy to be discontinued.
- Chronic pulmonary embolism in patients with pulmonary hypertension and cor pulmonale.

Endoluminal grafts

Principle

A combination of a stent with prosthetic graft positioned with a delivery system.

Uses

- Exclusion of A.A.A
- Support of the intima of an artery after balloon dilatation in some cases.

Thrombolytic therapy

The aim is to inject certain substances which activate the fibrinolytic system leading to dissolution of fresh thrombi in the arterial or venous system, Ideally the fibrinolytic agent should be given as a regional therapy directed into the thrombus via an endovascular catheter.

Indications

- Acute deep vein thrombosis.
- Acute thrombotic ischaemia. in this situation after dissolution of the thrombus if there is a stenotic segment, it can be treated by balloon angioplasty and/or stenting.
- Acute coronary occlusion.
- Occlusion of vascular grafts.
- Restoring the patency of recently occluded central venous catheters.
- Pulmonary embolism.
- Acute embolic ischaemia.

The procedure is better done in an ICU under close observation.

The main contraindications include: active bleeding, recent stroke, major operations within two weeks, infected bypass grafts and/or infective endocarditis.

The main agents used are streptokinase, urokinase and recombinant tissue plasminogen activators.

VENOUS DISORDERS

Venous Thrombosis General Principles

Types:

- Superficial venous thrombosis.
- Deep venous thrombosis occurring mainly in the calf or iliofemoral veins.

Less common sites are the inferior vena cava, subclavian, axillary or portal veins.

Superficial Thrombophlebitis



Aetiology

- Varicose veins.
- Veins cannulated for I.V. infusion.
- After injection of irritant drugs, e.g., diazepam.
- Associated with:
 - Buerger's disease (thrombophlebitis migrans).
 - Polycythaemia.
 - Polyarteritis.
 - Visceral cancers (it may be the earliest sign of malignancy).
- Idiopathic.



Clinical Picture

1. The vein becomes red, painful and cord like.
2. Mild pyrexia may be present.



Complications

- Infection spread: rapid upward spread occurs with the danger of extension to the deep veins through the communicating veins. In this condition, proximal ligation and disconnection should be done.
- Pulmonary Embolism: As the thrombus is adherent to the vein wall because of inflammation.



Treatment

- Compression by elastic stocking or compression bandage.
 - Anti-inflammatory drugs, e.g. aspirin.
- These are usually enough for the majority of cases. The following are resorted to under special circumstances.
- Antibiotics only if there is evidence of infection.
 - Anticoagulant therapy (heparin and warfarin) is given in severe progressive cases (ascending thrombophlebitis), and if associated DVT is suspected.
 - Surgery: Prophylactic sapheno-femoral or sapheno-popliteal disconnection is done if the process of thrombosis is ascending towards the junction with the deep system.

Factors that help venous return from the lower limbs

- The muscle pump which requires strong muscles and intact deep fascia.
- Transmitted arterial pulsations to the venae comitantes.
- The unidirectional valves.
- The negative intrathoracic pressure.

Pathogenesis Virchow's triad: 3V

- (Vessel Wall) Damage to the endothelial lining of the vein wall due to:
 - Trauma to the vein wall, e.g., during pelvic operations.
 - Inflammatory process near the vein, e.g., pelvic sepsis.
- Venous stasis: (Velocity)
 - Prolonged confinement to bed, long trips, or casts.
 - Congestive heart failure.
 - Venous compression by tumours or a pregnant uterus.
- Hypercoagulability of blood due to: (Viscosity)
 - Deficiency of antithrombin III, proteins S and C, macroglobulins or antitrypsin.
 - Polycythaemia.
 - Factor V Leiden gene defect or activated protein C resistance.
 - Dysfibrinogenemia.

Deep Venous Thrombosis (DVT)



The true incidence of DVT is not exactly known as many cases pass unnoticed. The incidence varies greatly in different countries, which may be due to racial and seasonal variations.

Predisposing factor:
All the factors mentioned before under Virchow's triad. Added to these are obesity, old age. Oral contraceptive intake, previous DVT, malignancy and major trauma.

DVT is common post operative complication. Its clinical picture varies greatly from silent cases to severe local and systemic symptoms.



Pathophysiology

- The process usually starts in the calf venous sinuses or in the iliac and femoral veins by adherence of platelets to the endothelial surface.
- More platelets then adhere. Fibrin and RBCs are deposited as layers in-between the platelets giving laminated appearance.
- When the vein is totally occluded, non-adherent, jelly-like propagating thrombus spreads up the vessel as far as the next major tributary. The thrombus is loosely attached and can be easily detached leading to pulmonary embolism.
- Later, the thrombus becomes adherent to the vein wall. It then organizes and contracts producing destruction of the valves and luminal narrowing, which are responsible for the eventual development of the post-phlebitic limb syndrome.
- Later on, the processes of fibrinolysis and phagocytosis start and lead to recanalization of the vein but the valves are permanently destroyed.



Clinical Picture

The classical group presents by a triad:

- **Pain:** There is usually aching discomfort and tightness in the involved calf or thigh.
- **Swelling:** This is the most reliable physical sign. It is evidenced by measuring the difference in the circumference between both limbs. In calf thrombosis, the swelling is limited to the foot and ankle, in femoral thrombosis the swelling involves the calf and lower part of the thigh, while in ilio-femoral thrombosis there is massive swelling affecting the whole limb.
- **Tenderness:** is present on grasping the affected calf or thigh or on compressing the muscles against the bones. Dorsiflexion of the foot causes pain in the calf (Homan's sign). However, this sign is not reliable.

The complication group

- **Phlegmasia alba dolens:** Massive iliofemoral DVT may be associated with severe arterial spasm. The limb becomes pale, white, and massively swollen with absent peripheral pulses.
- **Phlegmasia cerulea dolens:** Massive iliofemoral DVT may cause severe congestion. The whole lower limb is massively swollen and bluish. If not promptly treated it may lead to venous gangrene.
- **Venous gangrene.**
- **Pulmonary embolism.**

The asymptomatic group

Silent DVT is a frequent occurrence. There are no local symptoms and the patient may present later with either pulmonary embolism or with the manifestations of a post-phlebitic limb. However, it may be suspected by the presence of unexplained rise of temperature or pulse rate.



Investigations

- **Doppler Ultrasound:** It is a simple bed-side and rapid method and is accurate in 80-85% of cases. Ultrasound is insensitive in calf vein thrombosis.
- **Duplex ultrasound scan:** This method combines Doppler ultrasound flow analysis with ultrasound imaging. Its sensitivity and specificity reach 90-100% and most duplex errors are in below knee veins. Therefore, duplex scanning is considered the standard test for diagnosis of DVT.
- **Enhanced helical CT:** can show the thrombus even in small veins.



Differential Diagnosis

Doppler & Duplex Scan Findings In Cases of DVT		
	Normal veins	DVT
Vein diameter	Normal	Dilated vein
Blood flow	Spontaneous	Poor
Vein compressibility	Normal	Poor
Echogenic material in lumen	None	Present
Distal compression	Augments blood flow	Poor augmentation
Blood flow with respiration	Phasic flow	Loss of phasic flow

- **Sprain:** of calf muscles and haematoma.
- **Rupture of plantaris tendon:** Both previous conditions occur during exercise and can produce a swollen painful calf which is usually difficult to differentiate from DVT. Duplex scan is needed to establish the diagnosis.
- **Ruptured Baker's cyst.**
- **Lymphatic obstruction:** The swelling is chronic and usually non-pitting.
- **Cellulitis:** There are systemic symptoms and signs of inflammation and the leg is red, hot and tender.



Prevention

of postoperative DVT

Physical measures to reduce venous stasis:

- Early ambulation after operations and active leg exercises while in bed.
- Adequate postoperative hydration.

The above measures are routine precautions for all postoperative patients.

- Elastic stocking support especially in the elderly.
- intermittent pneumatic compression: Intraoperative and postoperative of the lower limbs.

Prophylactic anticoagulants

• Indications:

High-risk cases for development of DVT:

- o History of DVT or pulmonary embolism.
- o Major surgery, particularly cancer operations.
- o Females on contraceptive pills.
- o Elderly patients.
- o Obesity.

• Contraindications:

Prophylactic heparinization should not be used in case if a large raw area is left after surgery.



As clinical examination is not reliable (incorrect in about 50% of cases), investigations should be done before starting treatment.

• Methods:

- o Low-dose heparin (Mini-Heparin) 5000 IU subcutaneously 2 hours before operation and then every 12 hours until the patient is ambulant (5-7 days) proved very effective in lowering the incidence of DVT by 50%.
- o Low molecular weight heparin is more popular because it is given once daily and has lower risk of bleeding, making it more suitable for use at home.



Treatment (once DVT occurred)

1. Bed rest and elevation of the lower limb

- Strict bed rest with the feet elevated 15-20 degrees above the level of the heart. This reduces oedema and pain and increases venous return so reduces further thrombosis.
- Application of an elastic bandage or stocking will also help venous return.

• Thrombi usually take 7-10 days to become adherent to the vein wall, the patient should be kept in bed for this period. Usually the swelling, pain, and tenderness would have resolved by this time. Graduated ambulation with elastic support is then allowed but standing and sitting with the legs dependent are forbidden because the accompanying rise in venous pressure aggravates oedema and discomfort. These measures are continued for 3 to 6 months until recanalization and collateralization develop.

2. Anticoagulant therapy

Heparin

Mode of action:

- Enhances the activity of the naturally occurring antithrombin III. The antithrombin III-heparin complex neutralizes the effect of thrombin.
- Neutralizes factors IX, X, and XI.
- It may also have a mild thrombolytic effect as it can cause mild activation of fibrinolysis.

Administration:

Heparin is given until all signs of active thrombosis subside which is clinically known by disappearance of pain and tenderness. This usually takes 7-10 days.

Differences Between Unfractionated & Low Molecular Weight Heparins		
	Unfractionated heparin	Low molecular weight heparin
Mode of action	Antithrombin III	Anti factor Xa
Half life	1.5 - 2 hours	12 hours
Route of administration	I.V.	Subcutaneous
Monitoring	Activated partial thromboplastin time	Not required . if needed, measure level of Xa
Incidence of heparin induced thrombocytopenia	1-5%	<2%
Hospitalization	The patient should be hospitalized	May be taken at home

The objectives of treatment are:

- Prevention of formation of new thrombi.
- Prevention of pulmonary embolization.
- To minimize venous valves damage.

Fractionated (low molecular weight) heparin

- o Tinzoparin 175 IU/Kg/24 hours subcutaneously.
- o Enoxaparin 1 mg/Kg/12 hours subcutaneously.

Unfractionated heparin

- o Continuous IV infusion: This is the ideal method, but it requires hospitalization and a pump which controls the dose per hour. A bolus dose of 5000 IU is given IV and then a Dextrose 5% drip containing heparin is infused at a rate of 20-30 IU of heparin/kg/hour. This usually means 1000-2000 IU/hour for an adult. The dose is monitored by measuring the activated partial thromboplastin time (APTT) which should be kept between 2-2.5 times the control value. A more practical but less accurate method of monitoring is to do clotting time.
- o Bolus therapy 5000 IU are administered intravenously every 4-6 hours, the APTT is performed one hour before every injection until the dose is adjusted.

Complications

1. Bleeding :
due to over dosage.

Clinically: manifests by subcutaneous bruises, epistaxis, bleeding gums, hematuria, or gastro-intestinal bleeding.

Prevention: by proper control of the dose by repeated APTT.

Treatment: by giving protamine sulphate 1 mg IV for every 100 IU of heparin.

2. Failure to respond to heparin (heparin resistance): It may be mild and requires only increasing the dose of heparin.

3. Heparin induced thrombocytopenia occurs in 1-5% of patients usually in the second week of therapy. If the platelet count drops below 100,000/ul, heparin should be stopped.

Oral anticoagulants (coumarin derivatives)

warfarin (The most commonly used)

Mode of action:

These drugs block the synthesis of at least 4 vitamin K dependent clotting factors (prothrombin and factors VII, IX and X)¹⁹⁷² For this reason, its anticoagulant effect is delayed.

Administration:

- Basal level of prothrombin time (PT) and concentration are estimated prior to start of warfarin.
 - An initial dose of 10 mg warfarin is followed by 5 mg daily dose.
 - Discontinue heparin after 3 days of overlap treatment.
 - Five days after the start of warfarin the prothrombin time (PT) and concentration (PC) are measured and the dose is adjusted to reach the therapeutic goal of PC 30-40%.
 - PT and PC should be repeated every 2 weeks during the course of treatment.
- The dose is adjusted accordingly. These tests are more accurately expressed as the international normalized ratio (INR), which should be prolonged to between 2.5-3.5 times the control value.

Duration: Oral anticoagulants are given for 3-6 months which is the time needed for recanalization and collateralization. In some patients who are liable to rethrombosis, warfarin is given indefinitely.



- The use of subcutaneous low molecular weight heparin is less likely to cause bleeding.
- Oral anticoagulants inhibit the synthesis of protein C and S which have an anticoagulant effect and so there is a period of relative hypercoagulability during the first few days of warfarin therapy. This period should be covered by continuing heparin therapy together with the warfarin for the first three days.

Complications

- Bleeding is the main problem and is treated by lowering the dose and giving 10-20 mg vitamin K intravenously. In cases of severe bleeding fresh frozen plasma is given.
- Interaction may occur between oral anticoagulants and other drugs like aspirin and non-steroidal anti-inflammatory agents.

Contraindications to anticoagulant therapy

• Absolute:

- o Trauma to or recent operation on the brain or spinal cord.
- o Haemorrhagic diathesis.

• Relative:

- o Major visceral injury.
- o Major acute fractures.
- o History of cerebral haemorrhage.
- o Hypertension where the blood pressure is higher than 180/120 mm Hg.
- o Wide raw area.
- o Peptic ulcer.

3. Thrombolytic therapy

Indications: Patients with severe DVT (including phlegmasia alba dolens and phlegmasia cerulea dolens) are better treated by thrombolytic therapy.

Mechanism of Action: Fibrinolytic activators dissolve fresh thrombi and produce rapid clearance of the occluded veins and may preserve the competence and function of venous valves so They limit the development of the postphlebitic syndrome.

Examples of fibrinolytic activators

include streptokinase, urokinase and tissue plasminogen activator (TPA).

The last drug is prepared by recombinant gene technology. The effect of these drugs is at its best if they are given in the first 3 days of thrombosis, after that they have no advantage over heparin.

Complications

- Streptokinase may lead to allergic reactions.
- Severe bleeding. These drugs are contraindicated in old age, ulcer and history of haemorrhagic diathesis.

4. Insertion of inferior vena cava filter

Indications:

- Progressive thrombosis or recurrent pulmonary embolism despite anticoagulant therapy.
- If there is a contraindication to the use of anticoagulants.

Procedure:

Under local anesthesia and radiographic control the filter is placed by the percutaneous or open exposure method through the jugular vein. The filter is placed in an infrarenal position. Complications occur in less than 5% of cases and include haemorrhage, injury of IVC, perforation to the aorta, and migration.

Axillary-subclavian DVT

Incidence: 2-3% of all cases of DVT.

Aetiology:

- Spontaneous (primary): Excessive muscle activity of the upper extremity (sports or occupational) due to compression of the vein in the costoclavicular space. A cervical rib is a predisposing factor.
- Secondary to:
 - Central venous catheters.
 - Metastatic tumours in the axilla.
 - IV chemotherapy or parenteral hyperalimentation through upper limb veins.

Clinical features:

- Pain and swelling of the entire upper extremity.
- Prominent venous collaterals over the shoulder and anterior chest wall.
- The limb may be cyanosed.

Treatment:

Similar to that of lower limb DVT. Resection of a cervical rib may be required if it kinks the vein.

Pulmonary Embolism (PE)

+ Aetiology

All the predisposing factors leading to DVT are important in PE.

🧬 Pathophysiology

The effects of PE are relatively insignificant until more than 25% of the pulmonary artery circulation is occluded. Cardiac output remains normal until pulmonary artery occlusion exceeds 50% (massive embolism).

Underlying cardiac or respiratory insufficiency contributes to premature failure of cardiac output. Decreased pulmonary blood flow results in:

- Reduced cardiac output (CO) leading to systemic hypotension and shock.
- High V/Q ratio due to reduced circulation in areas of normal ventilation.
- Myocardial hypoxia.
- Pulmonary infarction occurs in less than 10% of PE cases. Its development depends on completeness of occlusion and effectiveness of collateral circulation.

👤 Clinical Picture

- The clinical picture of pulmonary embolism is determined by the site where the embolus impacts. Accordingly, three types of PE are recognized:
 - **Small emboli:** are impacted in the peripheral arterioles. They are usually silent. Recurrent small emboli lead to pulmonary hypertension.
 - **Medium sized emboli:** lodge in the branches of the pulmonary arteries and result in pulmonary infarction.
 - **Large emboli:** are impacted either in the main pulmonary artery or in one of its major branches causing massive pulmonary embolism. There is severe precordial pain, tightness in the chest, marked dyspnoea, severe hypotension and marked tachycardia. Sudden death may occur.
- In some patients with pulmonary embolism there is no clinical evidence of deep vein thrombosis in the lower limbs.

🏥 Differential Diagnosis

- Pneumonia.
- Congestive heart failure.
- Myocardial infarction.
- ARDS.



- Around 2-3% of all hospital mortalities are due, wholly or in part, to pulmonary embolism. Pulmonary emboli originate from thrombi in the venous circulation. They are, therefore, referred to as thromboemboli.
- Unexplained dyspnoea or heart failure appearing in a hospitalized patient is very suggestive of pulmonary embolism.



Investigations

To Diagnose:

- **D-dimer:** This is a degradation product of fibrin. It is elevated with DVT and PE. A normal value excludes pulmonary embolism.
- **Blood gases:** Low PaO₂ and normal PaCO₂.
- **ECG:** to differentiate PE from myocardial infarction.
- **Chest X-ray:** normal in 50% of cases or may show the following:
 - Prominent pulmonary artery (hilar shadow).
 - Diminished vascular markings in the affected area of lung.
 - Enlarged right ventricle.
 - Small pleural effusion.
- **Ventilation perfusion pulmonary isotope scan V/Q scan :** shows lung areas that are ventilated but not perfused.
- **CT pulmonary angiography is the most popular imaging study:** This is because it is a good diagnostic tool and is non-invasive as there is no need for vascular catheterization.
- **Pulmonary arteriography:** Visualization of an intraluminal filling defect or abrupt vessel cutoff is diagnostic. Pulmonary angiography though diagnostic is dangerous in shocked patients. Also, microemboli might be overlooked with pulmonary angiography.

Of the cause:

- Duplex scan: of lower limb veins to detect occult venous thrombosis.

Treatment

- Patients with pulmonary embolism are classified into three classes
 - **Mild pulmonary embolism (low risk):** there is no right ventricular strain, patients are treated by heparinization for 10 days then by warfarin.
 - **Submassive:** There is right ventricular strain detected by echocardiography and increased troponin level.
 - **Massive:** There is persistent hypotension <90 mm Hg for 15 minutes.
- Submassive and massive cases, patients should be admitted to ICU for:
 - Oxygen inhalation.
 - Cardiac supports as dobutamine or epinephrine.
 - Pulmonary catheter directed thrombolytic therapy.
 - pulmonary embolectomy: is indicated If there is continuous deterioration after thrombolytic therapy.

Vena caval interruption: The objective is immediate prevention of recurrent PE. This is indicated in the following:

- When anticoagulant therapy is contraindicated because of major bleeding complications.
- Recurrent embolism in spite of anticoagulant therapy.
- First PE in high risk patients.
- Following pulmonary embolism.

Varicose Veins Of Lower Limbs

A common condition where the superficial veins are dilated, elongated & tortuous.



Types

There are two types of the disease

Primary varicose veins: (where the cause is not known)

Theories are:

- **The weak wall theory:** assumes an inherited weakness of the vein wall, producing venous dilatation even with normal pressures. Secondary valvular incompetence occurs.
- **The congenital valvular incompetence theory:** A positive family history can only be found in 50% of cases of primary varicose veins.

Aggravating factors:

- Female sex: The condition is commoner in females.
- High parity.
- Occupations: requiring prolonged standing.
- Marked obesity.
- Oestrogens intake: e.g., contraceptive pills.

Secondary varicose veins: These develop secondary to:

- **Deep venous thrombosis:** This is the usual cause of secondary varicose veins. Recanalization of the thrombosed deep veins leaves the valves of the perforating veins incompetent leading to reflux of blood. This puts unusually high strain on the superficial veins which have little external support, so they progressively dilate.
- **Arterio-venous fistula:** Varicose veins are prominent with traumatic or congenital (Klippel-Trenaunay syndrome) A-V fistulae. Here, the arterial pressure is transmitted to the deep system leading to valvular incompetence and reflux of blood to the superficial veins which dilate and become varicose.
- **Pelvic tumors and pregnancy:** due to compression of the pelvic veins



Clinical Picture



Symptoms

- Cosmetic disfigurement.
 - aching, and discomfort of the leg usually described as dull, heavy, bursing sensation usually at the end of the day or on prolonged standing and is relieved by elevating the limbs.
- Symptoms are prominent in the secondary type.
- Night cramps.
 - Mild swelling occurs at the end of the day, particularly with secondary VV.
 - Pigmentation. This is more in secondary varicose veins due to ooze of blood in the subcutaneous tissue and deposition of haemosiderin.
 - Itching.
 - Ulceration is more with secondary varicose veins.

Post-phlebitic syndrome

- Sometimes there is history of DVT.
- Lower leg eczema and pigmentahon.
- Leg pain on standing relieved by elevation.
- Lower leg chronic ulcer
- Leg oedema on standing and by end of day.
- Small varicosities of irregular distribution.

Special tests

• Trendelenburg test

o Aim:

- Detection of saphenofemoral incompetence.
- Detection of incompetence of communicating veins.

• Multiple tourniquet test:

Rapid filling in any segment indicates the site of regurgitation.

• Perthe's test & Modified Perthe's test:

o Aim: Detection of the patency of the deep venous system.



Signs

The aim of examination is to get the following information:

- The anatomical distribution of the veins.
- Are the varicosities primary or secondary?
- Competency of the saphenofemoral junction & other communicating veins.
- The condition of the deep system of veins.
- The presence of complications.

The patient should be examined while standing and exposed up to the umbilicus. The following signs are to be noticed:

- Varicose veins appear as elongated, dilated and tortuous veins.
- The veins may belong to the long or short saphenous system in case of primary varicose veins or may be arranged haphazardly in case of secondary varicose veins.
- Presence of veins crossing the suprapubic region denotes secondary varicose veins caused by narrowing of the external iliac vein.
- Presence of complications as oedema, eczema, liposclerosis or ulceration is in favour of secondary varicose veins.
- Palpation of a thrill over the veins denotes an arterio-venous fistula.
- Palpation of a thrill over the saphenofemoral junction on cough denotes an incompetent saphenofemoral junction.

Differences between primary and secondary varicose veins.

	Primary VV	Secondary VV
Aetiology	Idiopathic	Secondary to previous DVT, A-V fistula, pelvic tumors or pregnancy
Pain	Slight or absent	Marked
Distribution	Along the long or short saphenous veins	Haphazard. Veins crossing the groin may be seen
Complications	Minimal or absent	Oedema, pigmentation, eczema, & frequent features



Complications

Complications are more frequent in association with secondary varicose veins. These are mainly manifestations of the post-phlebotic syndrome. The varicose veins themselves tend to produce no or minimal complications.

- Oedema.
- Subcutaneous bruises due to rupture of small veins as a result of venous hypertension.
- Itching, dermatitis and eczema are due to deposition of haemosiderin in the subcutaneous tissues.
- Liposclerosis: The extravasated fibrinogen leads to fibrous tissue formation. The soft supple subcutaneous fat is replaced by tough fibrous tissue.
- Recurrent superficial thrombophlebitis.
- Venous ulceration.
- The combination of the above-mentioned complications, together with chronic leg pain, constitute the «post-phlebotic» syndrome as they usually follow previous DVT with incompetent perforating veins.
- Haemorrhage may occur due to rupture of a varicose vein especially when the overlying skin is thin. It can be initially stopped by elevation and compression bandage. Later treatment is by injection sclerotherapy.



Investigations

- The aim is detection of the sites of incompetent communicating veins and to verify the patency of the deep veins. It should be noted that most cases of primary varicose veins require no investigations because careful clinical assessment provides enough information.

- **Duplex ultrasound:** is the investigation of choice. It serves accurate detection of incompetent perforators, and detection of accompanying deep vein thrombosis in suspected cases.



Treatment

Primary varicose veins

1. Conservative treatment: Patients with minor cosmetic or spider varicosities are treated by reassurance and elastic stocking.

2. Injection sclerotherapy:

• Indications:

- o Small unsightly veins.
- o Localized dilated superficial veins.
- o Incompetent lower leg perforators.
- o Recurrent or persistent veins after operation.

- **Principle:** Sclerosants like polidocanol should be injected in an empty vein to induce injury of the endothelial layer of intima. Compression is then applied. The walls of the vein will adhere together, thus permanent occlusion of the vein occurs.

• Complications:

- o Extravasation of sclerosant material under the skin leads to sloughing of the skin and poor cosmetic results.
- o Deep venous thrombosis may occur if a large amount of the sclerosant material reaches the deep system undiluted so no more than 1 ml is injected at any point and no injection is done for veins above the knee.

• Technique:

- o A venous tourniquet is applied over the thigh while the patient is standing and the site of the vein is identified and marked on the skin by a skin marker.
- o The patient is asked to lie down and a thin hypodermic needle is inserted into the vein.
- o The limb is then elevated by an assistant and one ml of the sclerosant material is injected.
- o An elastic bandage is then applied over the whole leg from the toes to the knee and is left for 4-6 weeks.
- o Immediately after the application of the bandage, the patient is instructed to walk for a long distance in order to flush any amount of sclerosant that might have reached the deep veins.

3. Surgery:

- Patients with clear evidence of long or short saphenous incompetence or a combination of the two should be treated by saphenofemoral or saphenopopliteal disconnection combined with stripping.
- Ligation of the saphenofemoral junction is called Trendelenburg operation. The saphenous vein should be disconnected flush with the femoral or popliteal vein and all tributaries near their termination must be ligated and divided to prevent recurrence.
- The long saphenous vein is then stripped from above the knee to the groin. The leg part of the vein is better not stripped to avoid injury of the saphenous nerve. The short saphenous is stripped from the lateral malleolus to the popliteal region.
- Recently, obliteration of the sapheno-femoral junction and medium sized incompetent perforators is being performed by endovenous laser or radiofrequency current.

Secondary varicose veins

- **Post phlebitic:** (i.e. following deep venous thrombosis) The majority of cases are treated conservatively by elastic stockings. Rarely the varicosities are large enough to require active treatment. In these cases, verify that the deep system is recanalized clinically and by duplex, then treat as primary VV.
- **Varicose veins complicating AV fistula:** Surgical treatment of the fistula is usually followed by marked regression of varicosities. If residual veins remain, treat as primary varicose veins.
- **Varicose veins occurring during pregnancy:** A complete elastic stocking from the toes up to the groin is applied all through the period of pregnancy. After delivery an residual veins are treated as primary varicose veins.

Venous Ulcers



Aetiology

- Venous ulcers usually occur in a postphlebotic limb due to previous deep vein thrombosis.
- A much less common cause is arterio-venous fistulas.
- Rarely the ulcer is a complication of primary varicose veins. In this case, it is usually transient and heals rapidly.



Pathophysiology

- Recanalization of DVT is commonly followed by dysfunction of the valves of both the deep veins and the perforating veins. Furthermore, it may produce narrowing of a large deep vein.
- As the skin of the lower part of the leg is drained directly by the perforators, which drain into the deep system, it follows that incompetence of these perforators leads to high venous pressure in the skin particularly that lying on the medial side of the lower third of the leg «ulcer bearing» area Or «gaiter» area
- The combination of venous hypertension, eczema and liposclerosis (fibrosis of subcutaneous fat) will eventually lead to ulceration after minor trauma.
- Local hypoxia due to venous stasis and impaired nutrition lead to the liberation of free oxygen radicals which are toxic to the tissues.



Clinical Picture

- The clinical features of a venous ulcer are characteristic.
- It is usually situated in the ulcer bearing area or around the medial malleolus. The skin surrounding the ulcer shows pigmentation, eczema with itching marks and induration.
- Secondary varicose veins may be present.
- The ulcer takes a long time to heal and is liable to break down after healing.



Treatment

Conservative treatment is indicated for all cases.

- Elevation of the foot in bed.
- Ulcer care. Moist saline dressings twice weekly.
- Elastic stocking or crepe bandage. Compression is the most important item in conservative treatment.
- Some medications are said to accelerate ulcer healing:
 - Pentoxifylline (Trental).
 - Prostaglandin E1 analogue.
 - Diosmin.

Conservative treatment is usually successful in allowing the ulcer to heal within a few weeks. The problem is that, once the patients return to normal activity, most ulcers will recur. If there is evidence of incompetent perforators in the leg, they can be either injected or excised.

Previous operations as open or endoscopic subfacial ligation of perforators are not commonly performed nowadays.



- Venous ulcers constitute 90% of chronic leg ulcers.
- Topical antibiotics should not be used as they may aggravate the condition by inducing an allergic reaction.

Lymphatic Disorders

Acute Lymphangitis



Aetiology

strept. (commonest).



Clinical Picture

- Fever, rigors & general constitutional disturbances may be very severe.
- Locally, there is pain, edema & red tender streaks. The regional LNs are enlarged & tender.



Treatment

1. TTT of the cause.
2. Antibiotics, especially penicillin & broad-spectrum antibiotics.
3. Local rest of the affected part & local heat to help resolution.
4. If suppuration occurs, it needs an incision.

Lymphadenopathy

The causes of LN enlargement are:

1. Inflammatory:

a. Acute:

- I. Septic lymphadenitis (non specific).
- II. Infectious mononucleosis (specific).

b. Chronic:

- I. Non-specific.
- II. Specific:
 1. Bacterial: T.B., \$.
 2. Parasitic: Filarial.
 3. Viral: Lymphogranuloma inguinale, cat scratch disease, AIDS.
 4. Protozoal: Toxoplasma.

2. Lymphomas:

- a. Hodgkin's lymphoma.
- b. Non-Hodgkin's lymphoma.

3. Blood Diseases:

- a. Acute leukemia.
- b. Chronic myeloid leukemia.
- c. Chronic lymphatic leukemia.

4. Lipoidosis.

5. Metastases.

6. Collagen diseases.

Acute Septic Lymphadenitis



Clinical Picture

1. The picture of the causative lesion.
2. General constitutional manifestations.
3. Locally the nodes are enlarged, red, hot, tender, firm or soft & if suppuration occurs fluctuation will be evident.



Treatment

- TTT of the causative focus.
- General rest and antibiotics.
- If an abscess forms, incision & drainage.



Complications

1. Spread to more proximal lymph nodes.
2. Spread to nearby tissues.
3. Suppuration (abscess formation).

Chronic Non-Specific Lymphadenitis



Clinical Picture

- Common in the submandibular and inguinal LNs.
- The nodes are slightly enlarged, mobile, slightly tender & firm in consistency.



Treatment

- This is directed to the original focus. The nodes need no ttt.
- If it persists for > 3 or 4 m, TB must be excluded.

T.B Lymphadenitis

Types:

1. lymph borne (common in young).
2. blood borne (common in elderly).

Lymph-borne (fibrocasseous) type (the commonest)

Sites:

1. Cervical LN groups: (commonest) the organisms reach from the tonsils.

2. The mediastinal & axillary groups (esp. in children): are $\pm$ T.B lesions in the lung.

3. Abdominal LNs (children & adolescents): the organisms from the ingested milk pass through the lacteals to reach the LNs without affecting the intestinal wall.

4. In people above 50, a common finding in x-ray is multiple mottled calcific shadows of mesenteric gls that are considered to be old T.B nodes healed by fibrosis & calcification (tabes mesentrica).



Pathophysiology

1. The organisms reach the LNs by afferent lymphatics thus reaching the capsule 1st → T.B periadenitis → matting of the nodes.
2. Then the cortex will be affected & finally the medulla.
3. Multiple tubercles will form, coalesce together & may caseate & break down → cold abscess that may rupture through the skin → T.B. sinus or ulcer.



Complications

1. Caseation & Cold abscess formation: which may burrow through the deep fascia or an overlying muscle → bilocular (collar stud abscess). 2nd infection may occur → difficult ttt.
2. Cold abscess: is actually neither cold (clinically warm) nor abscess (contents are not pus but caseating material).
3. Sinus formation: with a thin cyanotic undermined edges & thin serous discharge.
4. Spread to other groups of L.N.s: if the disease is left untreated.



Clinical Picture

Lymph born type	Blood born type
1. Common. 2. Usually in children 3. Local: a. Localized lymphadenopathy affecting the upper deep cervical LNs. b. Variable consistency: • Early → Firm • Caseation → Cystic • Calcification → Hard c. LNs. are matted together. L.N.s. may be arranged in beaded cords due to thickening of connecting lymphatics. 4. Caseation → cold abscess → T.B. sinus.	1. Rare. 2. Usually in old. 3. Local: a. Generalized lymphadenopathy b. Firm in consistency. c. LNs are discrete. As the pt is usually an adult of fair resistance, there is no breaking down, caseation or cold abscess formation. 4. No caseation. No cold abscess.



Investigations

1. Chest x-ray.
2. Tuberculin test: good -ve indicator.
3. Biopsy from the nodes: will establish the diagnosis.
4. Aspiration of cold abscess & ex. of the pus microscopically & guinea pig inoculation.
5. Smears from a sinus & ex. for tuberculous organisms.



A. Tuberculous lymphadenitis before caseation:

1. Good diet & vitamins.
2. At least 2 antituberculous drugs (Rifampicin + INH) for at least 9 m.
3. Surgical excision: for single group of LNs showing no response to medical ttt after 6 m.

B. Cold abscess:

1. Antituberculous drugs.

2. Aspiration & injection of streptomycin solution.

The rules of aspiration should be followed to avoid sinus formation:

- a. The needle is inserted in a healthy part of the skin away from the abscess.
- b. The site of puncture should be in a non-dependent part.
- c. The needle should also pass in a Valvular manner (i.e the points of entry through the skin and the abscess cavity should not be opposite to each other).
- d. The aspiration usually needs repetition every few days until the abscess dries up.

3. Incision: Indicated in the following conditions:

- a. 2ry infection.
- b. If the abscess is imminent to rupture.

4. Excision: if the nodes need excision !!.

C. TTT of a tuberculous sinus:

1. Antituberculous treatment.
2. Dressing with streptomycin powder every 3 days until it closes.
3. Excision with the underlying nodes, if resistant to conservative measures.

Lymphomas

Definition:

- Malignant neoplasm that arises in LNs or extra nodal lymphoid tissue.
- The main types are: 1. Hodgkin's disease. 2. Non-Hodgkin's lymphoma. 3. Burkitt's lymphoma.

Hodgkin's disease

More common

Non-Hodgkin's lymphoma

Less common

Pathology

Macroscopically

- It usually starts in the cervical lymph nodes.
- The affected nodes are enlarged, discrete, rubbery & have a pink color.
- Usually there is a large lymph node in the center with smaller LNs around it.
- There may be extra nodal affection of the spleen, liver or bone marrow.
- Pleomorphism.
- The characteristic «Reed Sternberg cells». These are giant cells that have an even number of nuclei (2 or 4), which are arranged in a mirror image manner.

LNs:

- 1) Markedly enlarged, amalgamated.
- 2) Early invasion of the surrounding structures.
- 3) Variable consistency but usually hard.

Classification

Histological types:

(In descending order of prognosis)

- 1) Lymphocyte predominance. (Best prognosis)
- 2) Nodular sclerosis. (The commonest variety)
- 3) Mixed cellularity.
- 4) Lymphocyte depletion.

Based on the cell of origin:

1. B Cell lymphoma is further classified into:
 - a. Small cell lymphoma.
 - b. Large cell lymphoma.
 - c. Mixed small and larger cell lymphoma.
 - d. Immunoblastic lymphoma.
2. T cell lymphoma.
3. Lymphoblastic lymphoma.
4. Histiocytic lymphoma.

In each type the cells may be arranged in a nodular or a diffuse pattern.

Staging

Ann Arbor staging system:

- **Stage I:** Single involved lymph node group (I), or a single extra lymphatic site (IE).
- **Stage II:** Two or more involved lymph node groups limited to one side of the diaphragm or a solitary extra lymphatic site and one or more lymph node areas on the same side of the diaphragm (IIE).
- **Stage III:** Involvement of both sides of the diaphragm.
- **Stage IV:** Extra lymphatic spread including liver, lung, bone marrow, skin and gut.

All stages are subdivided into either:

1. No systemic symptoms
2. One or more of the 3 systemic symptoms; fever, night sweats & weight loss of > 10% in 6 months.

C/P

- Incidence:
 - Race: More in Caucasians.
 - Age: Two age peaks, between 15-35 years, and above 50 years.
- Painless progressive enlargement of cervical LNs.
- With progress of the disease, other LN groups are affected. These nodes are enlarged, discrete, non-tender & rubbery in consistency.
- Splenomegaly (Toffee-almond appearance) & hepatomegaly may be present.
- Some pts exhibit systemic manifestations in the form of fever, night sweats, weight loss, pruritis, anemia & jaundice. Sometimes a characteristic intermittent fever. It lasts for 2-3 weeks followed by a remission for few weeks (Pel-Ebstein fever).
- Incidence: common with the following conditions: Sjogren's disease, Immune deficiency, SLE.
- LN enlargement: Progression of LN enlargement does not follow an orderly anatomical pattern as in Hodgkin's disease.
NHL is more likely extra nodal than Hodgkin's lymphoma e.g.:
 - a. Gastric lymphoma produces manifestations that are similar to carcinoma.
 - b. Intestinal lymphomas may produce intestinal obstruction, bleeding, or perforation.
- The disease is likely to be disseminated at the time of presentation.

Investigations

A. Laboratory:

1. Blood picture: May show: Anemia, eosinophilia or lymphopenia.
2. High ESR.
3. Alkaline phosphatase: is elevated in cases with bone or liver involvement.

B. Radiological investigation:

1. Chest X-ray & CT scan: allow detection of intrathoracic disease.
2. Abd. US & CT scan: have replaced lymphangiography for detection of para-aortic LN deposits.

C. Surgical investigation:

1. LN biopsy: The corner stone of diagnosis.
2. Staging laparotomy:
 - Indications: Stages IA, IB & IIA
 - The operation includes:
 - a. Splenectomy: For staging & avoids the need of its irradiation
 - b. Biopsy of both liver lobes.
 - c. Biopsy of all intra-abdominal lymph node groups which are marked by metal clips to help future localization by the radiotherapist.
 - d. Bone marrow biopsy from the iliac crest.
 - e. In females the ovaries and Fallopian tubes are fixed in the middle line behind the uterus to guard them against irradiation.
- Staging laparotomy is not preferred in many centers because:
 - a. The high accuracy of CT scan, and the increasing availability of MRI.
 - b. The risk of overwhelming post-splenectomy infection (OPSI).

TTT

- Stage IA, IB & IIA are treated by radiotherapy.
- Stage IIB is treated by radiotherapy & 6 cycles of combination chemotherapy.
- Stage III and IV are treated by 12 cycles of combination chemotherapy MOPP (Mustin, Oncovin, Procarbazine & Prednisone).

Prognosis:

An average 80% 5 years survival is obtained with proper treatment.

1. Radiotherapy & combination chemotherapy according to the stage of the disease.
2. Surgery:

Indication:

- a. Gastric or intestinal lymphoma.
- b. Staging laparotomy.

III- Burkitt's Lymphoma



Aetiology

Unknown may be related to infection with Epstein Barr (EB) virus (Malaria may have a role).



Clinical Picture

1. Common below the age of 12 years, in western Africa.
2. The usual presentation is painless progressively enlarging jaw swelling.
3. It may affect kidneys, retroperitoneal tissues, ovaries, long bones & CNS.



Treatment

Chemotherapy.

- Further cycles are needed to prevent recurrence

N.B. Pain in Hodgkin lymphoma is late and may occur with drinking alcohol.

Hodgkin's disease

1. The neck is the preferred site for LN biopsy.
2. In LN biopsy, we should take the whole LN without rough manipulation to preserve its architecture.
3. Splenectomy is important to spare the kidney and lung the hazards of radiation injury.
4. Radiotherapy is used as a supplement to control bulky cervical LN.
5. 5-year survival rate is 100% with proper treatment of stage IA with lymphocytic predominance.

Non-Hodgkin's disease

1. It has higher incidence with AIDS.
2. It may be related to infection with HTLV-1.
3. B cell lymphoma is the commonest type.
4. LN enlargement is similar to that of Hodgkin's disease.
5. Staging laparotomy is rarely indicated.
6. Mycosis fungoides is a variant of NHL in which skin eruption is the 1st of the disease manifestation.
7. Surgery is followed by radiotherapy and chemotherapy.
8. Gastric lymphoma has a better prognosis than gastric adenocarcinoma.

Chronic Lymphatic Obstruction (Lymphedema)



Definition

Hypertrophic condition of the skin & SC tissues caused by chronic lymphatic obstruction.

Sites: Limbs (L.L lymphedema is the most common type / post-mastectomy lymphedema of the U.L. is another common type), scrotum, external genitalia & rarely breasts.



Aetiology

A. 1ry Lymphedema:

Due to congenital malformations of the lymphatic vessels:

	Lymphedema congenita	Lymphedema Praecox	Lymphedema Tarda
Incidence	10%	80%	10%
Age	At or within 1 y of birth	Usually at adolescence	After 35 years
Sex	M > F	F > M	M = F
Site	Commonly bilateral & involves the whole leg	Commonly unilateral & below the knee	Usually unilateral

B. 2ry Lymphedema: Common

- Post-traumatic:
 - Injuries as circumferential skin loss.
 - Operations as Extensive block dissection of regional nodes.
 - Burns at the site of LNs.
 - Irradiation of the regional LNs.
- Parasitic: Filariasis is the commonest cause in Egypt.
- Post-Inflammatory:
 - Chronic (recurrent acute) lymphangitis.
 - Chronic specific lymphangitis e.g. T.B.
- Neoplastic:
 - Primary affection as lymphomas.
 - Secondary affection by metastases.



Pathophysiology

Pt with lymphatic obstruction will suffer from 2 problems:

- Lymph stagnation → accumulation of large amount of protein rich fluids (irritant) in tissues → FB reaction → fibrosis in the SC tissues → more obstruction.
- Recurrent attacks of lymphangitis → more obstruction → vicious circle.

Pathological changes:

- Swelling: at first due to accumulation of fluids & later due to fibrous tissue replacing the SC fat.
- Edema: pitting then later, non pitting (fibrosis hardens the skin).
- The skin changes: negligible in 1^{ry} lymphedema but in long standing cases, there is thickening & hyperkeratosis. Lymphatic vesicles may appear in the skin. In severe cases, the skin develops huge bulges (elephantiasis).



Differential Diagnosis

Other causes of chronic diffuse limb swelling: postphlebotic limb, elephantiasis neurofibromatosis & congenital A-V fistula (local gigantism).



Clinical Picture

1. Type of pt: it differs acc. to history of the cause e.g. in case of filarial lymphedema: adult pt from an endemic area (e.g. Giza, Imbaba, Rasheed, Damietta, Sharkaya and Assyot) complaining of progressive leg swelling with exacerbations & partial remission.

2. During exacerbations: (Attacks of streptococcal lymphangitis) the limb shows red tender streaks traveling to draining LNs.

3. The swelling: is hard non-pitting with a crease in front of the ankle.

4. In late cases:

- The skin may show blebs, which may rupture → superficial ulcers oozing lymph (lymphorrhea)
- Skin is dark, thick with multiple deep furrows, thick callosities & warty over growths (Elephantiasis).

5. The inguinal LNs: may be enlarged, firm & tender (chronic septic lymphadenitis) or may form a soft lobulated mass (varicose LNs).

6. Other filarial manifestations: as lymphedema of scrotum, filarial funiculo-epididymitis, chylocele (rupture of lymphatics in tunica vaginalis), chylous ascitis & chylothorax.



Investigations

- Mid-Night blood film: for microfilaria.
- Intra dermal tests.



Treatment



Medical

• **Indication:**

Mild & moderate cases.

• **Method:**

- Limb hygiene.
- Elevation, massage & exercise.
- Elastic stockings
- Diuretics.
- Long acting penicillin 1,200,000 U every 3w to stop the recurrent attacks of lymphangitis.
- Active filarial infection is treated by diethyl carbamazine.



Surgical

I. Physiological Operations:

1. Enteromesenteric bridge operation: A segment of the ileum with its mesentery is separated & is brought under the inguinal ligament. The mucosa of the bowel is removed and the opened bowel is sutured over the cut LNs.

2. Omental flap operation: A segment of the greater omentum is mobilized with intact blood supply and is transferred into the lower limb to increase the lymphatic drainage.

3. Microlymphatico-venous anastomosis: The dilated obstructed lymphatic trunks are anastomosed to nearby veins.

4. Microlymphatic transfer operation: healthy lymphatic trunks are harvested from the normal limb & anastomosed to bypass the obstructed lymphatics.

5. Lympho-venous anastomosis: A LN is bisected & anastomosed to a nearby vein.

II. Excisional operations:

1. Charles operation: Excision of skin & SC tissues + Covering the limb by split skin graft

2. Sistrunk operation: Excision of an ellipse of skin & SC tissues + Closure of the defect. This decreases the bulk of the limb at one side. This can be repeated on the other side at later date.

III. Physiological and excisional operations:

Thompson operation (Swiss roll operation): of excision of SC tissue and then implantation of a shaved flap of skin bet. the ms near the deep lymphatic vessels so that the physiological drainage may be improved.

Filarial Lymphedema



Introduction

- This is the commonest cause of lymphedema in tropics.
- It occurs where culex pipiens mosquito (intermediate host) exists.
- Very heavy and repeated infestation by mosquito bites is essential for pathology to start.



Aetiology

Wuchereria Bancrofti.

Life cycle:

- In man (definitive host) the mature worms live in the lymphatics & LNs.
- The microfilaria are laid by the female & then circulate in the peripheral bl. at night. They have a nocturnal periodicity (between 10 pm. - 2 am. with the peak round midnight).
- The microfilaria are sucked by the mosquito culex pipiens (intermediate host). In the body of mosquito, the microfilaria passes to stage of embryofilaria (infective stage) where it is injected into the body of pt by bite of mosquito.
- The embryofilaria mature to adult worms.



Pathophysiology

- Adult worms live in the cortex of lymph nodes obstructing the outflow of lymph.
- This produces cystic enlargement of lymph node, which is given the name of varicose gland.

Role of filaria worms is just to start attacks of streptococcal-lymphangitis by causing lymphostasis. Big number of worms is essential. Once lymphangitis starts, the disease continues automatically like any lymphedema, whether worms are living or dead.

Neck

Anatomical divisions of the neck

Triangles:

1. Posterior.
2. Anterior.

Further divided into:
Submandibular,
Carotid & Muscular
triangles.

Compartments:

The pre-vertebral fascia
divides the neck into 2
compartments:

1. The muscular
compartment posteriorly.
2. The visceral
compartment anteriorly.

Branchial Cyst

Aetiology

Origin:

- In embryo, there are 5 grooves on each side of neck called branchial clefts (like fish gills). Normally, the 2nd arch grows rapidly covering the 3rd & 4th arches then it fuses with the neck. The space bet. the 2nd arch & the rest of arches turn into a cervical sinus which disappears soon.

- 1st cleft persists as external auditory meatus.
- Other 4 clefts disappear normally.

2nd cleft is the commonest to persist & produce 2 anomalies:

1. Branchial fistula: If the 2nd arch doesn't completely fuse with the neck.
2. Branchial cyst: If the cervical sinus persists.

Incidence: Congenital but presents in childhood or later.

Pathophysiology

o **Lining:** Sq. epithelium, surrounded by lymphoid tissue, which explains its frequent inflammation

o **Content:** Mucus rich in cholesterol crystals (rectangular with notched corner).

Clinical Picture

o Site:

- a. Upper part of side of neck: below angle of mandible deep to upper 1/3 of sternomastoid protruding beneath its anterior border.
- b. On contracting sternomastoid, it seems to bulge out.

o Characters:

- a. Usually starts in the late childhood.
- b. Moderate size (5 cm diameter), smooth, globular, tense cyst, well defined & opaque.

Differential Diagnosis

From other swellings in the lateral aspect of the neck.

- It is differentiated from cold abscess by finding cholesterol crystals in the aspirate.

Complications

Frequent inflammation as its lining is surrounded by lymphoid tissue.

Treatment

Complete excision through transverse incision.

Branchial Fistula



Aetiology

as before...



Pathophysiology

- It's usually congenital but occasionally acquired 2ry to ruptured infected branchial cyst.
- The track is lined by sq. epith. & extends up to the sidewall of the nasopharynx (fossa of russenmullar).
- **Course:** it passes bet. the CCA bifurcation deep to the post. belly of digastric ms & superficial to the hypoglossal nerve.



Clinical Picture

- **At birth:** it presents as pin point opening at the ant. border of the lower 1/3 of sternomastoid ms.
- The opening discharges mucus, if infected → pus.
- It may be occasionally confused with a T.B sinus.



Treatment

Excision of the whole track through multiple transverse neck incisions:

- Small one around the fistula opening & the other at higher levels below the jaw.
- Ureteric catheter is introduced into the track to facilitate its identification during surgery.

Cystic Hygroma (Cavernous Lymphangioma)



Aetiology

- Normal development: the lymphatic system develops by the coalescence of multiple small lymph vesicles. A large accumulation of these lymph vesicles is present lat. to the jugular vein & called jugular lymph sacs.
 - Abnormal development: if some vesicles fail to join the lymphatic system, they become sequestered & form cystic hygroma.
- Site: neck (the commonest), cheek, tongue, axilla, groin, mediastinum.



Pathophysiology

- It consists of multiple cysts with larger ones near the surface while smaller ones are deep & infiltrating tissue planes.
- Each cyst is lined by endothelial cells & contains clear lymph.



Clinical Picture

(at birth or within the 1st few years of life):

- Painless swelling presents usually in lower part of the post. triangle.
- It's bluish (thin overlying skin), translucent, soft, partially compressible & ↑ in size with coughing & crying.
- It may ↑ rapidly in size & interfere with respiration.



Treatment

Excision at the age of 3 years. This could be facilitated by preoperative injection of boiling water in the swelling to induce fibrosis to make it smaller.

D.D. Of A Mass In The Neck

Mid line swellings

Solid swellings:

1. Submental LN enlargement.
2. Nodule in the isthmus of thyroid gland.

Cystic swellings:

1. Cold abscess. «Rare in the midline»
2. Dermoid cyst: Sublingual or suprasternal.
3. Subhyoid bursitis: It is a rare, tender, oval swelling which lies transversely beneath the hyoid bone. It moves up and down with deglutition & with protrusion of the tongue.
4. Laryngocele: It occurs in musicians playing with air-blown instruments.
 - It is a herniation of laryngeal mucosa through the thyrohyoid membrane.
 - The swelling is resonant, compressible & increases in size with coughing or blowing.
5. Cystadenoma of the thyroid isthmus
6. Thyroglossal cyst.

Swellings in the Digastric «Submandibular» triangle

1. Enlarged submandibular LNs: multiple, can be rolled over the edge of the mandible & can't be felt in the floor of the mouth.
2. Enlarged submandibular salivary gland.

Swellings in the Carotid triangle

Solid swellings:

1. Enlarged upper deep cervical LNs.
2. The upper part of an enlarged lateral lobe of the thyroid gland.
3. Carotid body tumor:



Pathophysiology

It is a rare slowly growing malignant tumor arising from the chemoreceptors present at the bifurcation of the carotid artery.



Clinical Picture

- It's usually smooth but may be lobular.
- The swelling moves from side to side but not vertically. It may be pulsating.



Investigations

Angiography can prove the diagnosis.



Treatment

- By excision of the tumor with preservation of the ICA.
- If preservation of the ICA is impossible, it should be replaced by a graft.

Cystic swellings:

1. Cold abscess.
2. Branchial cyst.
3. Aneurysm of the carotid artery.

o Diagnosis depends on Pt's age, Clinical Course, Site & Consistency (solid/cystic).

o It should be noted that the most common swellings of the neck are swellings of LNs & thyroid, followed by those of the salivary glands.

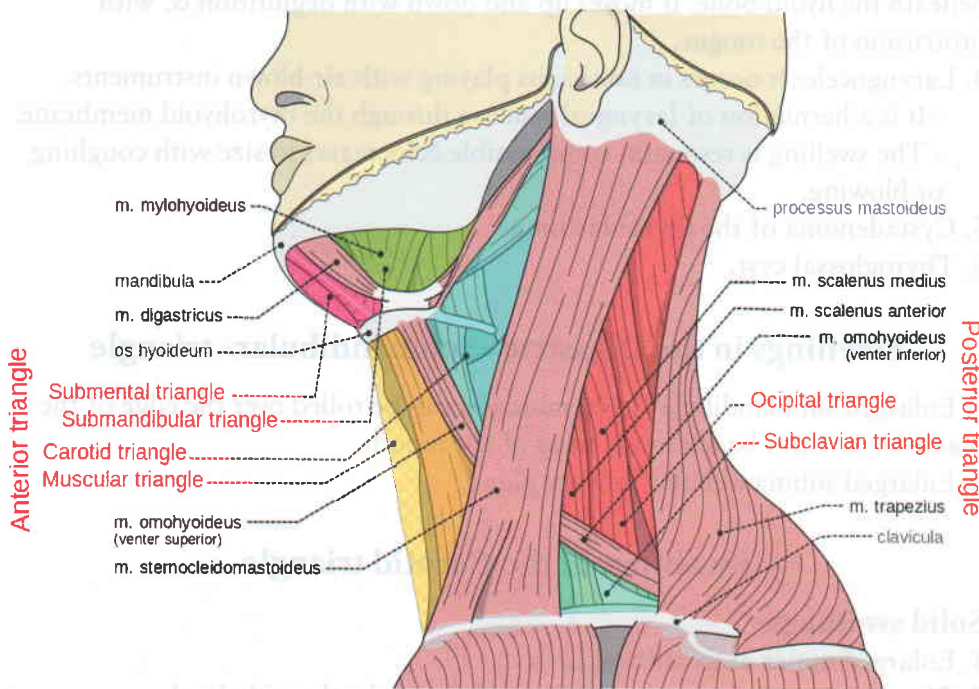
Swellings in the Posterior triangle

Solid swellings:

1. Enlarged LNs.
2. Neurofibroma arising from the brachial plexus.
3. Cervical rib.

Cystic swellings:

1. Cystic hygroma
2. Pharyngeal pouch
3. Cold abscess
4. Pneumatocele: This is a cystic swelling in the supraclavicular region, which is resonant & compressible. It is due to herniation of the pleura in the base of the neck.



• In addition, it should be noted that swellings of the skin and subcutaneous tissue are common in the neck region and should be put in mind such as lipoma, sebaceous cysts & hemangiomas, which are particularly common in the neck and face regions.

• Neck swellings with expansile impulse on cough & compressible: Laryngocele, Pharyngeal pouch & Pneumatocele.

Sternomastoid Tumor & Congenital Torticollis

Normal development : the sternomastoid muscle develops through union of three somites , each with its own blood supply

+ Aetiology

Abnormal development

Interruption of the bl. supply to the central somite of sternomastoid ms → ms infarction which becomes swollen (the name sternomastoid tumor). After a while the infarcted part will be replaced by fibrous tissue that will contract → congenital torticollis.

Clinical Picture

- At birth (or shortly afterwards) there is a painless firm swelling at the middle of sternomastoid ms.
- Later when torticollis develops the head will be tilted to the side of the lesion with the face looking to the opposite side.
- If the condition is neglected → facial asymmetry.



Differential Diagnosis

From 'wry neck' which is fibrositis causing spasm of the sternomastoid ms. This condition is temporary lasting for a day or two & responds to anti-inflammatory drugs.



Treatment

- **Early after birth:** Attempt to prevent the development of the deformity by stretching & splinting of the neck may be done.
- **Established deformity:** Division of the sternomastoid at its lower part is done.

Neck Injuries



Aetiology

Blunt & Penetrating trauma.



Pathophysiology

- Blunt trauma usually causes fracture of the spine & only in severe injuries it may be associated with visceral & vessel injuries.
- Penetrating injuries deep to the platysma are usually associated with injury to viscera & vessels.



Complications

1. Fracture of the cervical spine → quadriplegia or paraplegia.
2. Cranial nerve & Brachial plexus injury.
3. Injury to the larynx or trachea → respiratory obstruction, aphonia or hoarseness of voice. Laryngeal stenosis may be a late complication.
4. Arterial injury → hge which may be severe enough to cause shock. It may also lead to interruption of the bl. supply to the brain → infarction, neurological deficit & expanding hematoma.
5. A vein injury in addition to hge → air embolism.
6. Injury to esophagus → leakage & severe infection in the neck which may extend the mediastinum.



Clinical Picture

Of complications.



Investigations

- Plain X-Ray & Angiography are very useful to detect fractures & vessel injury. However, in cases with severe bleeding, immediate exploration is necessary.
- Duplex U/S, CT & MRI brain.
- Laryngoscopy, Bronchoscopy.
- Gastrographin swallow.



Treatment

1. **Life-saving measures:** 1^{ry} & 2^{ry} surveys.
2. **Definitive ttt:**
 - Visceral & vascular injuries: Exploration & repair.
 - TTT of spinal injuries.

Cellulites Of The Neck

Superficial cellulites: «As cellulites anywhere. If above hyoid → Sudden asphyxia especially in children from sudden edema of the glottis»

Deep cellulites (Ludwig's angina): It's usually results from streptococcal infection.



Pathophysiology

- It is a clinical condition characterized by inflammatory swelling of both the submandibular region & the mouth cavity
- Infection is deep to the deep fascia & can spread to the glottis → edema & asphyxia



Clinical Picture

- Painful, red, warm, tender submandibular swelling.
- The overlaying skin is edematous.
- Tongue swelling pushing it up & forwards.



Treatment

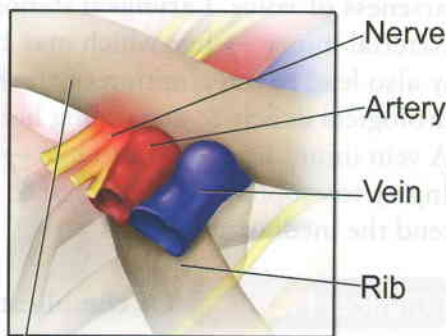
Antibiotics & drainage through an incision beneath the jaw deepened to permit submandibular gland displacement. Mylohyoid is divided to allow for adequate drainage.

Thoracic Outlet \$



Aetiology

1. Cervical rib which may be complete or incomplete. It extends from the C7 vertebra to the 1st rib.
2. A fibrous band extending from an incomplete cervical rib to the 1st rib.
3. A tight scalene muscle.
4. Post fixation of the brachial plexus. In this case the lower root of the brachial plexus arises from T2 instead of T1 thus this nerve becomes excessively bent over the 1st rib.



Clavicle (collarbone)

- Brachial plexus & subclavian artery pass to the upper limb through narrow triangle in the base of the neck.
- This triangle is made of scalenus ant. ms anteriorly, scalenus medius ms posteriorly & 1st rib inferiorly.
- At this narrow space compression of nerves and artery may occur.



Complications

- **Post-stenotic dilatation:** It is an aneurysmal dilatation, which may send a shower of emboli to the index & middle fingers, as they are the direct continuation of the brachial artery.
- **Compression of subclavain vein** → subclavain & axillary vein thrombosis & chronic venous insufficiency of upper limb. «Chronic heaviness & swelling»



Clinical Picture

- More commonly in females. About 20 years.
- Usually unilateral affecting the right arm (dominant arm).

H Symptoms

- **Arterial:** Intermittent claudication which appears with exercise .
- **Venous:** DVT and CVI .
- **Vasomotor:** Raynaud's phenomenon (Pallor followed by cyanosis then redness «Rebound Hyperemia») due to irritation of the sympathetic fibers.
- **Nervous:** tingling & numbness in the medial aspect of the forearm & the hand.
- **The lower trunk of Brachial plexus** is the main structure to be compressed.



Signs

- A bony swelling at the base of the posterior triangle (the bony cervical rib).
- Hypoesthesia in the medial forearm & hand and wasting of hand muscles may be noticed as well as weaker radial pulse on the affected side.
- Adson's test: The pt is asked to extend his head, look to the opposite side & take a deep breath. The examiner then palpates the radial pulse while the arm is pulled down. If the test is +ve, the pulse will be weaker. This test is not very accurate.



Differential Diagnosis

- Spondylosis.
- Carpal tunnel \$.
- Other causes of Raynaud's phenomenon .g. Raynaud disease, system lupus.



Investigations

- Plain X-ray to the neck may detect a bony cervical rib.
- Nerve conduction study between the neck & the forearm to detect delay in conduction. This is a useful investigation & can differentiate between thoracic outlet \$ & carpal tunnel \$.
- Subclavian angiography, Duplex U/S.



Treatment

- **No Symptoms:** No ttt.
- **Physiotherapy:** to strengthen the shoulder ms.
- **Surgery:** The following operations:
 1. Excision of a bony cervical rib. The rib should be excised with its periosteum.
 2. Incision of the scalenus anterior muscle (scaleneotomy).
 3. Excision of the first rib to relieve the compression inferiorly through a transaxillary approach (very efficient).

In many persons a cervical rib is detected by radiography, yet it is asymptomatic. Also in many pts who have the clinical picture of thoracic outlet \$, radiography does not reveal a cervical rib.

Radical Block Neck Dissection



Definition

Removal of all LNs on one side of the neck, in one mass (en block).

Indications:

- Operable primary malignancy of the head or neck, with palpable malignant cervical lymph node (The main indication)
- Prophylactic radical block neck dissection i.e. where cervical nodes are impalpable e.g. melanoma & carcinoma of the tongue as they have ↑↑ incidence of lymphatic spread.

Technical considerations:

- **Incisions:** the operation can be done through different incisions; the goblet incision is a popular one

Structure that are removed in radical block neck dissection:

1. All LNs on one side of the neck
2. Other structures are also removed because of their close proximity to the cervical nodes:
 - Sternomastoid muscle. - Carotid sheath. - Internal jugular vein.
 - Submandibular salivary gland. - Lower part of the parotid gland.

Variations of radical block neck dissection:

- **Suprahyoid block dissection:** it is restricted to the LNs above the level of the hyoid bone

It is indicated in lower lip carcinoma & in early cases of carcinoma of tip of tongue & floor of mouth. Submandibular glands are also removed because of their close proximity to these nodes.

- **Modified radical block dissection:** similar to radical block neck dissection but with preservation of IJV, sternomastoid & spinal accessory nerve. It's indicated in thyroid carcinoma & in bilateral block dissection as 1 vein (on the less affected side) is needed for the brain's venous drainage.

Retropharyngeal Abscess Acute retropharyngeal Abscess

This due to suppurative inflammation of retropharyngeal LNs which usually occurs as complication of tonsillitis. It is commonest in children under 4.



Clinical Picture

- The neck is held rigidly, saliva dripples from mouth and feeds are regurgitated.
- The posterior wall of pharynx is bulging and fluctuant swelling may be felt digitally.



Treatment

- Under general anaesthesia and using cuffed endotracheal tube, pair of forceps guided by the finger is thrust into abscess cavity. Pus is evacuated and antibiotic therapy is prescribed.



- In other malignancies with impalpable cervical nodes, follow up every 2 months & do radical dissection if they enlarge.
- In bilateral block dissection, preservation of 1 jugular vein on the less affected side is necessary.
- Carotid artery, Vagus nerve & Accessory nerve are preserved.

Pharyngeal Diverticulum {Pharyngeal Pouch}

- A protrusion of mucous membrane through the Killian's dehiscence (pulsion diverticulum) due to raised intrapharyngeal pressure, from failure of relaxation of the cricopharyngeus (achalasia) during contraction of the pharyngeal ms above it, in the act of swallowing.
- Once formed, the protrusion fills with food at every meal & progressively, increases in size displacing the esophageal opening. The pouch descends towards the mediastinum, but often turns outwards, usually to the lt to appear in the post. triangle of the neck.



Clinical Picture

- The pt is usually an elderly & the condition is twice as common in men as in women.
- It may give rise to symptoms identical to a foreign body in the throat.
- In long-standing cases there is history of dysphagia, recently aggravated and ass. with regurgitation of undigested food after meals, eructation of gas & a gurgling noise in the neck.
- Sometimes, there is an irritable cough from overflow of food into the larynx.
- In about 1/3 of cases, the pouch is large enough to form a visible swelling in the neck, which enlarges when the patient drinks and can be emptied by pressure.
- In advanced cases, progressive loss of weight occurs and may lead to cachexia.



Diagnosis

- Barium swallow may show a bulge in the posterior wall of the pharynx in early cases, but in late cases there is a flask shaped pouch sometimes with a fluid level.
- Esophagoscopy is not recommended as it may perforate the fundus of the pouch.
- Manometric studies may be helpful.



Treatment

- When the pouch is of a large size, surgery is recommended.
- The diverticulum is excised, its neck closed in 2 layers & a cricopharyngeal myotomy is performed.

Post-Cricoid Carcinoma



Incidence

More common in females (around age of 40 years).
May occur on top of Plummer Vinson's S.



Clinical Picture

1. Early symptoms: Throat Pain referred to: The side of the neck & Ear (stimulation of Arnold nerve).
2. Foul smelling of breath & loss of weight.
3. Dysphagia is late.

4. Ex.:

- Forward bulging of thyroid cartilage and trachea.
- The alae of thyroid cartilage may spread outwards.
- Loss of laryngeal click.



Investigations

Barium swallow, direct laryngoscopy & biopsy.



Treatment

1. Operable & fit pts:

- Total laryngopharyngectomy & block dissection of LNs.
- Replacement of pharynx by:
 - a. The stomach is mobilized & pulled up to the neck & anastomosed with the pharyngeal stump
 - b. Pectoralis major myocutaneous flap.
- The pt will be left by a tracheostomy.

2. Inoperable & unfit pts: Treated by external irradiation.

Tracheostomy

Indications :

1. Relieve obstruction of upper air passages due to
 - a. Foreign body
 - b. Edema of glottis .
 - c. Bilateral abductor paralysis of vocal cord .
 - d. Carcinoma of larynx
 - e. Injuries of larynx
 - f. Chronic stenosis following tuberculosis .
2. To allow assisted or positive pressure ventilation especially if needed for prolonged period as endotracheal tube cannot be left in place more than 10 days.

Post operative care :

1. Tube is connected to a ventilator .
2. Frequent suction of secretions .
3. Humidification of the inspired air to avoid dryness of air passages and secretions which become difficult to expel and aspirate.

Complications:

1. Early :

- a. Dryness of secretions and obstruction of tube .
- b. Damage of tracheal wall from prolonged pressure by cuff .
- c. Displacement of tube into tissues of neck .
- d. Surgical emphysema .
- e. Wound infection.

2. Late: Subglottic stenosis if tracheal incision involves first tracheal ring .

Face & Mouth

Congenital Anomalies Of The Face



Development of the face from 5 processes: Frontonasal process, 2 maxillary & 2 mandibular processes

The 1st 2 defects are the most common congenital anomalies of the head (1:700 live births).



Cleft lip may be associated with other congenital anomalies in up to 35 % of cases [Cleft palate, Velopharyngeal incompetence, Coloboma, Microphthalmia, Encephalocele, Ear tags & Torticollis].

1. Cleft lip
2. Cleft palate.
3. Preauricular sinus: due to imperfect fusion of the auricular tubercles. When occluded, a cyst is developed & might turn into an abscess, if bursts → Ulcer.
4. Sequestration dermoid cyst: at the lines of embryonic fusion. The most frequent is the ext. angular dermoid.
5. Pierre Robin S: Cleft palate, Receding mandible (micrognathia) & Posterior displacement of the tongue obstructing the oropharyngeal airway.
6. Mandibular prognathism (protrusion of the mandible).

Cleft Lip «Hare Lip»



Aetiology

- Failure of fusion between the median process of the frontonasal prominence & one (unilateral) or both (bilateral) maxillary processes.

Etioloaical factors:

1. Familial disease due to genetic susceptibility.
2. Consanguinity.
3. Prenatal exposure to alcohol, anticonvulsants, X-ray or virus infection (German measles in the 1st trimester).



Pathophysiology (Types)

1. Unilateral (most common on the lt side) or bilateral.
2. Complete (reaching the nostril floor) or Incomplete. With complete cleft lip, orbicularis oris is completely interrupted → Flaring & Flatness of the nares on the affected side.
3. Simple or complicated (if it is associated with cleft palate or alveolus).

Effects on function:

1. Cleft lip does not interfere with suckling, but may be associated with abnormal teeth growth.
2. Psychological upset of the parents.



Treatment

- Surgery is the only ttt.
- Timing: best performed at the age of 3-6 months (before teething).
- Perquisites: the infant should be at least 10 pounds in weight & the HB should be at least 10gm %
- Principles of the operation:
 1. Paring of the edges.
 2. The lip is separated from maxilla to make the lip lax.

3. Suturing the 3 layers of the lip (skin, ms & m.m) in a zigzag to avoid notching of the margin as the scar contracts.
4. In bilateral: The repair is done either as one stage or as 2-stage operation.

Cleft Palate (1/3 of all facial clefts)



Aetiology

- Incomplete fusion between the 2 palatal processes of the maxilla with the nasal septum & possibly the premaxilla (from the frontonasal process).
- PFs: See cleft lip.



Pathophysiology (Types)

1. Cleft uvula.
2. Cleft soft palate.
3. Cleft soft & hard palate (complete).
4. Complete cleft palate + one side of premaxilla (bipartite).
5. Complete cleft palate + both sides of premaxilla (tripartite).

Effects on function:

1. Impairment of normal suckling, due to inability to create a -ve intraoral pressure.
2. Regurgitation → aspiration pneumonia
3. Recurrent otitis media, which may lead to hearing loss. «Inadequate Eustachian tube aeration due to abnormal levator palati insertion»
4. Speech defects 2ry to:
 - a. Inadequate velopharyngeal mechanism opposite of valum (the ms of the soft palate) to separate the oral from the nasal cavity is impaired. Nasal tone due to naso-oral communication.
 - b. Hearing loss.
5. Interference with normal teething alignment.
6. Distorted facial growth.



Treatment

- **Timing of the operation:** at 12-18 months (before phonation).
- **Preoperative management:**
 1. Attention to feeding. As there is inefficient breast-feeding, a bottle with a large hole is used or spoon- feeding is used in an upright position.
 2. Prevention & ttt of chest infections.
- **Objectives of surgery:**
 1. Closure of oro-nasal communication.
 2. Achieving a competent velopharyngeal sphincter.
- **Principles of surgery :**
 1. Paring of the edges.
 2. Lateral relaxation incisions are needed.
 3. Suturing is done in 3 layers in the middle line (nasal mucosa, muscle layer then oral mucosa).
 4. Fracture of the pterygoid hamulus to relax the tensor palati.

• **Post-operative follow up:**

1. Speech therapy.
2. Tonsillectomy & adenoidectomy are better avoided.
3. Orthodontic ttt.

Maxillofacial Injuries

1. The maxillofacial region can be divided into 3 parts:

- The upper face: the frontal bone & frontal sinus.
- The mid-face: the nasal, ethmoid, zygomatic & maxillary bones.
- The lower face: the mandible.

2. Maxillofacial trauma is usually caused by: RTA, Fights & contact sports as soccer.

3. In ttt of pts with extensive maxillofacial injuries, the priorities are:

- a. Patent airway.
- b. Effective breathing.
- c. Control of he by direct pressure or ligation of the bleeding vs. if thereis shock → suspect ass. injuries as the isolated facial injuries rarely cause shock.

Fractures Of Maxilla



Aetiology

Usually 2ry to car accidents.



Clinical Picture

Pain, excess salivation, mal-occlusion, oedema, epistaxis, diplopia, swelling & crepitation.



Investigations

X-ray or CT scan.



Classification

• **Le fort I:**

- a. Transverse fracture above level of the teeth.
- b. TTT: intermaxillary fixation which in turn is fixed to the inf. orbital margin by wires.

• **Le fort II:**

- a. A pyramidal-shaped fracture traversing the base of nose through the posterior wall of maxillary antrum & across the orbit.
- b. TTT: intermaxillary fixation & fixed by wires to zygomatic process of frontal bone.

• **Le fort III:**

- a. There is a craniofacial dysjunction i.e. separation of facial bones from their cranial attachment.
- b. TTT: correction of nasal & zygomatic fractures. Then the same as in Le Fort II.

Fractures Of The Mandible



Aetiology

Traumatic (falls, kicks, fist blows, car accidents) or pathological.

Site:

- Fractures may occur in body (commonest), symphysis, angle, ramus, coronoid or alveolar process.
- Body injuries are usually close to the mental foramen where the bone is weakened by marked curvature & the deep canine socket. Bilateral injuries impair the airway as the digastric & geniohyoid pull the chin fragment & the attached tongue backwards.
- Angle fractures are minimally displaced as they're splinted by the masseter & pterygoid.



Clinical Picture



Symptoms

1. Pain on attempts to open the mandible.
2. Bl. stained saliva
3. They are usually compound into the mouth as the mucoperiosteum is firmly attached to the bone.
4. Impairment of speech & swallowing.
5. Anaesthesia of the lower lip (sometimes).



Signs

1. Coleman's sign: swelling & hematoma in the floor of the mouth.
2. There is irregularity of the line of the teeth.
3. Local tenderness & crepitus.



Investigations

Plain x-ray is diagnostic «Panoramic view», CT scan may be needed.



Treatment

A. 1st aid ttt: the jaw is supported, analgesics, antibiotics & oral hygiene to prevent infection.

B. Reduction & fixation:

- If there is displacement, reduction is done under anesthesia.
- Fixation for 3 weeks is done by:
 1. Interdental wire (the pt's jaws are wired together & a liquid diet is given) or Arch bars. This technique is appropriate for a majority of lower jaw fractures.
 2. Plates & Screws. More complicated fractures require ORIF by plates & screws.

Nose Fracture



Clinical Picture

- Pain, Swelling, Epistaxis & Crepitus.
- Plain X-Ray is diagnostic.



Treatment

- Reduce the fracture then fix by intranasal packing for 3 days with an external splint for 7 days.
- Neglected old fractures require osteotomies.

Fracture Zygoma



Clinical Picture

- Pain, Eyelid swelling, Flat cheek, Numbness in the cheek & upper teeth due to infraorbital N. injury.

Blowout fracture

- Depressed fracture of the orbital floor with herniation of some of the orbital contents into the maxillary sinus due to sudden ↑↑ in the intra-orbital pressure.
- A blow by a fist is a common cause.



Clinical Picture

- Diplopia, Limited up & down gaze ± Enophthalmos.

TMJ Dislocation



Aetiology

- Direct blows.
- Yawning.
- Wide opening of the mouth under anesthesia. «Commonly bilateral & affects mid. aged females»



Clinical Picture

- Pain, Dysarthria.
- Mouth is held open with fixed jaws.
- In unilateral cases, the chin is deviated to the opposite side.



Treatment

- Reduction (Better under anesthesia) by downward traction on the molars with the padded thumb together with upward rotation of the body with the outside fingers.
- In recurrent cases, excision of the meniscus is done.

Soft tissue injuries

- Facial nerve injures med. to midpupillary line need no ttt but if lat. requires repair under magnification.
- Parotid duct injures require end-to-end anastomosis over a small silastic catheter. Distal duct injuries require suturing of the proximal duct to the oral mucosa. Parotid gland injuries require drainage & skin suturing. Discharge stops in 3 weeks.
- Lips injures should be sutured in 3 layers with respect to the anatomical landmarks .
- Nasal injuries are sutured in 2 layers. Evacuation of septal hematoma to avoid saddle nose deformity.
- Intraoral injuries: edges are derided and loosely approximated.
- Lid injuries: Careful suturing of all lid layers. Repair of cut levator to avoid ptosis. Repair of tarsus. Lacrimal apparatus injury must be repaired to avoid epiphora & dacryocystitis.
- Ear injuries: Cutaneous perichondrial sutures for full thickness tears. Hematomas should be evacuated to avoid cauliflower ear.

Carcinoma of the face

Lip carcinoma

- Epithelioma (sq. cell carcinoma) is the commonest lip malignancy of the skin of the lip.

Aetiology

1. Prolonged exposure to the UV rays of the sun.
2. Continuous irritation & hyperplasia due to cigarette smoking or the use of hot tobacco pipes.

Pathophysiology

Microscopic

Sq. cell carcinoma which is usually well differentiated.

Gross

- a. The lower lip is more affected.
- b. The lesion usually starts as a nodule or erosion resisting ttt then later the typical ulcer becomes evident.
 - **Edge:** Raised everted.
 - **Base & Margin:** Indurated (induration extends beyond the edge).
- Spread to cervical LNs: (Stony hard & painless, at first mobile but later become fixed).
 - a. The central part of the lower lip drains to submental LNs.
 - b. The lateral parts drain to the submandibular LNs.
 - c. Later the upper deep cervical LNs are involved.



Dangerous area of the face

Definition

The area of the face between the lines passing from the outer canthus to the angle of mouth.

Importance

Infection in this area is liable to cause cavernous sinus thrombosis.

How does infection reach the cavernous sinus?

- The angular vein communicates with the ophthalmic veins which drain into the cavernous sinus.
- The anterior facial vein communicates with the cavernous sinus via an emissary vein.
- Pterygoid venous plexus is connected to the cavernous sinus by an emissary vein that passes through foramen ovale at the skull base.



Treatment

A. Surgery for 1ry tumor: excision with safety margin

- Lesions involving 1/3 of the lip → V shaped excision & 1ry suture in 3 layer: mucosa, ms & skin.
- Bigger lesions → plastic reconstruction.

B. Radiotherapy: good alternative as the SCC is radiosensitive.

C. LNs: if there are LN metastases → suprahyoid or complete block dissection.

Prognosis: Lip epithelioma has a better prognosis than cheek, tongue & floor of mouth.

Cystic Swellings Of The Face

1. External angular dermoid.
2. Meibomian cyst.
3. Mucocele of the lacrimal sac.
4. Meningocele.
5. Sebaceous cyst.

Items	Sebaceous cyst	Dermoid cyst
Cause	Retention	Sequestration, implantation or teratomatous
Site	Any site esp.: face, scalp & scrotum. Never in palm & sole.	Special site: line of fusion in face, midline & palm.
Shape	Hemispherical	Complete sphere
Attachment to skin	At one point	Not attached
Age	Adult	Dates since birth or childhood

Cystic Swellings Of The Floor Of The Mouth

[1] Mucous cysts of minor salivary glands.

[2] Ranula:

• **Def.:** retention cyst arising from a sublingual salivary glands. Saliva distends the cyst in the floor of the mouth.

• **Pathology:**

- The wall is composed of a thin fibrous capsule which is lined by macrophages.
- It may contain gelatinous material.
- If it's extended into the neck over the post. Margin of the mylohyoid (diaphragm of the mouth), it forms a plunging ranula.
- It may rupture but usually refills again.

• **C/P:** translucent, bluish painless swelling with prominent bl. vs running over its surface together with stretched submandibular duct.

• **TTT:** Excision is very difficult (as the wall is thin and adherent). Partial excision of the roof, the edges of which are sutured to the mucosa lining the floor of the mouth (marsupialization) is the ttt of choice (i.e. the floor of the ranula will form part of the floor of the mouth).

[3] Sublingual dermoid cyst: «Usually appears after puberty although congenital»

- It is a sequestration dermoid cyst occurring in the midline either supramylohyoid appearing in floor of mouth or inframylohyoid appearing in the neck.
- It is lined by sq. epithelium & contains sebaceous material.
- The wall is thick. It is not translucent (D.D. ranula).
- **TTT:** Supramylohyoid or small cysts are excised through the floor of the mouth. Inframyllohyoid or large cysts are excised through a curved submandibular incision.

The Tongue

Developmental Anomalies

A- Tongue Tie

- Due to short fibrous lingual frenulum. It causes impairment of tongue movements & possibly speech defects.



Treatment

Division of the frenulum below the undersurface of the tongue.

B- Macroglossia

- Persistent painless enlargement of the tongue.



Aetiology

• Congenital:

1. Cavernous hemangioma.
2. Congenital AV fistula.
3. Lymphangioma.
4. Neurofibromatosis.

• Acquired:

1. Cretinism.
2. Acromegaly.
3. Amyloidosis.

Tongue Injuries «In Epileptics, RTA»



Treatment

- Bleeding is arrested by hooking the tongue forward with a finger & compressing it against the mandible.
- Then the lacerations could be sutured in the operating room. Tracheostomy is needed in respiratory affection.

Chronic Superficial Glossitis



Aetiology

chronic irritation. It occurs in middle and old ages. PDF: (6 S)



Pathophysiology

1. The disease affects the anterior 2/3 of the tongue.
2. Epithelium overgrows → Leukoplakia (thickened, indurated & opaque areas) then it may shed over a considerable area → Red glazed tongue & finally Submucous fibrosis occurs → Ulcers, Fissures & Warty projections.
3. Chronic superficial glossitis is precancerous.



Treatment

1. Elimination of the irritating source.
2. Mouth wash.
3. In resistant cases, excision of the lesion.

Ulcers Of The Tongue

1. Dental ulcer:

- **Site:** Margin of the tongue.
- **Size:** Usually small.
- **Shape:** Circular with soft base & sloping margins.
- **Pain:** Usually present.
- **LN:** Chronic inflammatory enlargement.
- Simulates malignant ulcer in chronic cases so biopsy is needed to differentiate.
- **TTT:** Removal of the septic tooth or denture is followed by healing of the ulcer

2. Dyspeptic ulcer: «Apthous Ulcer»

- **Etiology:** unknown.
- **Site:** The sides of the tongue and its dorsum and inner surface of lip and cheek.
- **Size:** Small.
- **Surface:** Covered with white scabs.
- **Base:** Not indurated.
- **Edge:** Sloping.
- **Margin:** Hyperemic.
- **Number:** Multiple.
- **TTT:**
 - a. Of the cause. Paint ulcer with gentian violet.
 - b. Antiseptic mouth wash, alkaline lotion & anesthetic gel.

3. Neoplastic ulcers: Sq. cell carcinoma (discuss).

Carcinoma Of The Tongue

Incidence:

- **Age:** More above age of 60.
- **Sex:** M:F ratio = 10: 1. Now, this ratio is ↓↓. ↓↓ male incidence due to improved oral hygiene, efficient ttt of syphilis. ↑↑ female incidence due to ↑↑ smoking among females.
- **Geographical:** it has a high incidence in India due to ↑↑ tobacco chewers & use of spicy foods.



Aetiology

- **PFs:** Smoking, sepsis, spices, spirits, sharp teeth, clay pipe & bad oral hygiene.
- **Precancerous lesions are:**
 1. Syphilis.
 2. Dental ulcers.
 3. Chronic superficial glossitis.
 4. Papilloma of the tongue.
 5. Leukoplakia (Any white lesion that can't be rubbed off): It shows hyperkeratosis & possibly dysplasia.
 6. Erythroplakia: This is deep reddening with mucosal atrophy & leukoplakia.



Pathophysiology

Sq. cell carcinoma: (contributes in 90 % of cases)

- **Site:** Lateral margin of the anterior 2/3 (50%). Posterior 1/3 (20%). Less common sites are the ventral surface & the dorsal surface. The tip is rarely affected.



Gross

It is either :

1. Malignant ulcer: See carcinoma of the lip.
 2. Raised oval plaque.
 3. Hard submucosal nodule.
 4. Deep indurated fissure.
 5. Diffuse infiltrating tumor (woody tongue) is rare.
- The surrounding mucous membrane may show leukoplakia.



Microscopic

1. Sq. cell carcinoma (less differentiated than that of the lip). Post. 1/3 carcinoma is less differentiated
2. Adenocarcinoma from the minor salivary glands is rare & so is basal carcinoma.

TNM staging:

- | | |
|--------------------------------|--------------------------------------|
| • T0: No evidence of tumor. | • N0: No LNs. |
| • Tis: Carcinoma in situ. | • N1: Ipsilateral single node <3 cm. |
| • T1: <2cm. | • N2: LNs <6 cm. |
| • T2: 2-4 cm. | • N3: LNs > 6 cm. |
| • T3: >4cm. | |
| • T4: Involvement of the base. | • M0: No metastasis. |
| | • M1: Distant metastasis. |



Pathogenesis

1. Direct spread: To the floor of the mouth, mandible. Tumors of the posterior 1/3 spread to the tonsils, pharyngeal wall & larynx and may cross to the other side of the tongue.

2. Lymphatic spread: Unlike sq. cell carcinoma of the lip, carcinoma of the tongue disseminates early to the LNs of the neck:

- a. Tip: Spread from the tip of the tongue to the submental LNs then to both submandibular & to both upper deep cervical nodes on both sides.
- b. Anterior 2/3: Tumors of the lateral 1/3 disseminates to the ipsilateral submandibular & then to the upper deep cervical LNs. Those near the midline disseminate bilaterally.
- c. Posterior 1/3: Spread occurs directly to the upper deep cervical LNs.

3. Blood spread (Rare): This is more for tumors of the posterior third.



Clinical Picture

• **Age:** >60 years.

• **Sex:** more in males.

• **In early cases:** The patient may complain of :

1. Ulcer with indurated base and everted edges.
2. Deep indurated fissure.
3. Indurated, lobulated mass with overlying yellow submucosal necrosis.
4. Oval raised papillated plaque with overlying white keratin fleck.

• Late presentation:

1. Pain in tongue: The pain is referred to the ear through the chorda tympani via the auriculotemporal nerve. Pain, early due to infection, later due to lingual nerve infiltration.
2. Excessive salivation: which may be blood stained. The classic picture of tongue cancer is that of an old man sitting in the outpatient clinic with cotton wool in his ear & blood stained saliva dripping from the mouth.
3. Dysphagia: especially in posterior one third.
4. Feter: Due to necrosis and infection.
5. Inability to speak (dysarthria).
6. Secondaries in the neck.
7. Tongue fixation (Ankyloglossia): Extensive mouth floor infiltration.
8. Picture of complications.



Complications

1. Inhalation of necrotic tissue → bronchopneumonia.
2. Combined cancer cachexia & starvation due to pain & dysphagia.
3. Hge.
4. Spread.
5. Asphyxia may occur.



Investigations

1. Biopsy from the edge of the ulcer. Any resistant for treatment ulcer or fissure like lesion should be biopsied.
2. FNAC: of suspected cervical LNs.
3. CT of neck & mandible.



Treatment

[A] Radiotherapy: is indicated in:

Early cases. «T1, T2» - In tumors of posterior 1/3. - In inoperable cases.

[B] Surgical ttt: For operable cases it is indicated in:

1. Small Suspicious nodule. «T0, T1, T2»
2. Cancer on top of leukoplakia or Syphilis.
3. Tumor infiltrating the mandible (Bone).
4. Recurrence after irradiation.

• Surgical removal is as follows:

1. CiS: excision with 1 cm safety margin on the sides & 0.5 cm safety margin in depth.
2. Carcinoma of the anterior $\frac{2}{3}$: excision with 1.5 cm safety margin.
 - a. For the tip of tongue: V-shaped partial glossectomy is done.
 - b. For margin of the tongue: hemiglossectomy removing one half of the tongue is done.
 - c. Midline tumors: total glossectomy is done removing the whole tongue.
 - d. Tumors of posterior $\frac{1}{3}$: either total glossectomy (difficult) or external irradiation.
 - e. If the tumor infiltrates the mandible: Commando operation is done (combined mandibulectomy & neck dissection operation). Aiming at removing the lesion in continuity with lymphatic drainage. Preliminary tracheostomy is needed. Plastic reconstruction as pectoralis major myocutaneous flap is needed to close the defects.



- Irradiation can be performed by iridium or caesium needles or by external beam radiotherapy.
- Radiotherapy is avoiding the disfiguring side effects of surgery.
- Mucositis, dysphagia & osteo-radionecrosis are disadvantages of Radiotherapy.
- Preoperative care of teeth & oral hygiene. 4000 rad irradiation may be advised.
- T1 & T2 (Less than 4 cm) may equally benefit from surgery or radiotherapy.

[C] TTT of LN metastasis:

1. If LNs are not enlarged or are enlarged & not fixed: Prophylactic or curative complete block dissection is done. In bilateral cases, a complete dissection on the more affected side & selective dissection on the less affected side are done preserving the IJV is done.
2. If LNs are enlarged and fixed: Palliative deep X-ray therapy is done.

[D] Palliation is indicated for:

1. Unresectable 1st growth.
2. Fixed LNs in the neck.
3. Distant metastasis.

• Methods of palliation are:

1. Full dose of therapeutic irradiation is recommended.
2. Palliative resection of the 1st if possible may comfort the pt.
3. Analgesics, nasogastric feeding or tracheostomy may be required.
4. Chemotherapy.



Prognosis

(Prognostic Factors)

1. LN involvement is the most important prognostic index.
2. TNM staging.
3. The degree of tumor differentiation.
4. Combined surgery & radiotherapy improve prognosis.
5. Prognosis is better in females.

Cheek Carcinoma

- Sq. cell carcinoma is the commonest. Much less common is adenocarcinoma of the minor salivary gls.
- Predisposing & Premalignant conditions are similar to those of the tongue.
- Usually a malignant ulcer...
- External beam or interstitial radiation is the ttt. of choice.
- Surgery followed by reconstruction for recurrence or residual tumors.
- Neck dissection of LNs.

Swellings Related To The Jaw

1. Epulis (arising from the mucoperiosteum or gum margin):

- Fibrous, Myeloid, Granulomatous, Hemangiomas, Carcinomatous & Sarcomatous epulis

2. Odontomes (dental origin):

- Dental cyst, Dentigerous cyst & Adamantinoma.

3. Bone tumors:

- Ivory osteoma, Chondroma, Osteoclastoma, Maxillary antrum carcinoma, Fibrosarcoma, Osteosarcoma of the mandible & Secondaries.

Epulides

Any swelling situated on the gum, e.g.:

1. Fibrous epulis:

- It is an inflammatory hyperplasia of submucosa due to chronic irritation usually near the lower incisor. It is formed of fibrous tissue with spindle cells & plasma cells.
- The swelling is well defined, pedunculated & covered with intact mucous membrane.
- The swelling appears between 2 teeth.
- **TTT:** Extract a tooth on either side of the tumor & remove wedge of bone with its mucoperiosteum.

2. Granulomatous epulis:

- It consists of excessive granulation tissue due to chronic irritation either by tooth or irregular denture. The swelling is soft in consistency, bleeds easily on touch, red & not covered by epith..
- **TTT:** Of the cause. Cauterization either by diathermy or silver nitrate of this granulation tissue.

3. Hemangiomatous epulis:

- Submucous hemangioma of the gum. Blue in color, compressible, sessile, covered with intact mucous membrane. It is liable for severe bleeding.
- **TTT:** Injection of sclerosing material or surgical excision.

4. Myeloid epulis:

- It is an osteoclastoma arising in the inner osteoclastic layer of periosteum or alveolar margin & erupting through a tooth socket. It's commoner in the lower jaw.
- **Pathology:** Soft, lobulated, encapsulated swelling with cystic areas in cut sections. It consists of giant cells & spindle cells together with fibroblasts & fibrous tissue under the microscope.
- **Clinically** it forms a sessile smooth swelling covered with intact mm & fixed to underlying bone.
- Adjacent teeth are loosened. X-Ray shows bone eating.
- **TTT:** Wide excision with the part of the bone carrying the epulis.

5. Carcinomatous epulis.

- «Gum mucous membrane epithelioma. It's similar to mouth floor carcinoma in C/P & TTT. but radiotherapy has no role»

6. Sarcomatous epulis:

- It's a parosteal fibrosarcoma arising from the outer layer of periosteum. It may occur on top of a fibrous epulis.
- Tumor is sessile & fixed to the bone. X-Ray shows infiltration of the bones. It is treated by wide excision with the underlying bone up to hemimandibulectomy.

Odontomes

Tumors of the jaw arising from tissues involved in the development of teeth.

- The tooth has 2 different origins; 1st ectodermal from the dental plate & 2nd from mesodermal origin (mesodermal papilla). Between the developing teeth fragments of ectoderm may persist as the paradental debris of Malassez (this may be responsible for tumor formation later on).

- Odontomes may arise from ectodermal or mesodermal origin of a tooth or from both. Only those of epithelial origin occur in man (dental, dentigerous cysts & adamantinoma).

Dental Cyst

- o Cystic swelling related to the root of a normally erupted tooth (usually incisor tooth of the upper jaw).

- o It is due to proliferation of paradental epithelial debris due to chronic irritation by an infected tooth.

- o It is unilocular, slowly growing, lined with epithelium and filled with brownish fluid rich in cholesterol crystals.

Clinically:

- It occurs in adult life & is painless. The swelling is at first stony hard but later the bone may be thinned out so that «egg-shell crackling» can be felt. The tooth related to the swelling is grayish blue «Pulpless» & may be infected. X-Ray shows expansion of the jaw around a well-defined cavity containing no teeth or trabeculae.

TTT: Affected tooth extraction then incising the gum to open the cyst then cyst enucleation with removal of the lining membrane & fluid. The bone may be crushed to keep the shape of the alveolus.

Dentigerous Cyst

- o A cystic swelling related to the root of non-erupted tooth, which is contained into the cyst. It usually affects the 3rd molar tooth, premolar or canine.

- o It occurs in young age. It is commoner in the mandible near the angle than the upper jaw.

- o • The mandible is expanded uniformly and sometimes produces eggshell crackling. The cyst grows slowly.

- o It is lined with epithelium & filled with glory fluid around a non-erupted tooth.

X-Ray shows expanded jaw & clear cyst with a tooth in it.

TTT: Remove the roof of the cyst & the lining membrane.

- If the tooth is normally directed & expected to grow upwards normally, it is not removed.

- If the tooth is maldirected, this tooth is removed.

- Expanded jaw is crushed to restore its shape.

Adamantinoma «Eve's tumor/Ameloblastoma»

- o It may be a basal cell carcinoma arising from the epithelial debris of Malassez.

- o The tumor arises at the angle mandible.

- o It grows slowly forwards into the body of the mandible and upwards into the ascending ramus.

- o It consists of cystic and solid areas separated by fibrous and bony septa.

- o The cystic areas are lined sq. & basal cells & contain brownish mucoid fluid in the center. The solid areas are composed of fibrous tissue with particles of enamel.



Clinical Picture

- It is more in females.
- Age between 25-45 years.
- Pt presents with a painless, non-tender, lobulated swelling expanding the mandible on the outer side more than the inner.
- The consistency is bony hard, but eggshell crackling may be elicited.
- Mucous membrane may ulcerate by the tumor or the loosened teeth.
- LNs are enlarged with ulceration & 2ry infection.



Investigations

1. Plain X-ray shows expanding translucent shadow divided by numerous septa into equallobules (honey-comb appearance).
2. CT scan & MRI.
3. Biopsy.



Complications

1. Ulceration, infection, bleeding & falling of teeth.
2. Pathological fracture (rare).
3. It is a locally malignant lesion, so it is liable to recurrence after inappropriate surgery. Sometimes it turns malignant. «Sarcoma from fibrous element, Carcinoma from epithelial element»



Treatment

- Resection of tumor bearing segment of the jaw with a healthy margin on either sides up to hemimandibulectomy. The defect is repaired by bone graft.

Bone Tumors

As general

Giant Cell Lesions Of The Jaw

1. Giant cell granuloma:

- Affects the lower jaw more commonly.
- It arises above the symphysis menti as a soft reddish mass & grows backwards in the body of the mandible, but not upwards in the ascending ramus.

• **Cut section:** Lobulated tumor.

• **Histologically:** Giant cells, Spindle cells & Numerous blood vessels. Similar to myeloid epulis, brown hyperparathyroidism tumors.

• **C/P:** It is a painless, slowly growing tumor, expanding the jaw equally on both sides (due to presence of osteodasts). It may show eggshell crackling.

• **Investigations:**

- Plain X-ray: Expanding translucent shadow with unequal trabeculae (soap bubble appearance).
- CT, MRI, Biopsy.
- Serum Ca, PTH to exclude hyperparathyroidism.

- **TTT:** careful enucleation and gentle curettage. In large tumors, resection of the mandible is done.

2. Osteoclastoma: Rare & similar to the previous mentioned lesion, but it is malignant on histological ex..

3. Aneurysmal bone cyst: It forms a soft, sponge like tumor.

Carcinoma Of The Maxillary Antrum

- **Incidence:** Commonest malignant tumor in the maxilla.

- **Age:** 40 years.

- Equally affects both sexes.

- Chronic antral infection is a PDF. In most cases it's associated with 2ry nasal sinuses infection.

- The tumor is either columnar cell carcinoma from the antrum or sq. cell carcinoma (less commonly) from the hard palate, gums or tooth sockets.

- Invades adjacent structures. LN spread → Upper deep cervical, Retropharyngeal nodes. Blood spread is rare.

- **C/P:** «Depends on which wall of the maxillary antrum is involved»

1. Medial wall → unilateral nasal obstruction, bleeding from nose, epiphora (nasolacrimal duct obstruction) & foul nasal discharge.

2. Roof of the antrum → unilateral proptosis & diplopia.

3. Anterior & lateral walls → a swelling in cheek.

4. Floor, bulges in the roof of the oral cavity & may cause pain referred to the teeth.

5. Posterior wall leads to voice change, difficulty in breathing & post nasal discharge of pus & blood.

- **Investigations:**

1. Plain X-Ray:

- In early stages, it shows opacity & increase in the size of the antrum.

- Later, there is decalcification & bone erosion.

2. CT & MRI: Define the exact site, size & extent of the tumor.

3. Sinuscopy & Biopsy.

- **TTT:**

- 1. Early Cases:** Both surgery & radiotherapy produce equal results. Surgery is recommended with bone invasion as curability with radiotherapy is ↓↓ in these cases.

- Standard Operation: Total maxillectomy (Removal of the maxillary antrum with its wall). A synthetic prosthesis is fitted in place of the excised bone to avoid facial deformity.

- Radical block neck dissection in cervical LN involvement.

- 2. Advanced Cases:** A combination of radiotherapy & surgery is better than either alone. After a course of external irradiation, maxillectomy is done.

- 3. Recurrent cases:** Intra-cavitary irradiation by radium needles.

Hand Infections

General etiology:

- These infections are prevalent among manual workers.
- S.aureus is the causative agent in 90% of the cases.
- Reaches the infection site usually directly through minor injuries & punctures.

General C/P:

- FAHM
- Pain, Swelling.

General Investigations:

- FBS for recurrent infections.
- X-Ray for foreign bodies.

Principles for the ttt of the different types of hand infection:

1. Early administration of antibiotics effective against S. aureus.

«Flucloxacillin, Erythromycin, Amoxicillin/Clavulanic acid»

2. Hand elevation:

- ↓ Pain & edema.
- If there is marked swelling, the hand is bandaged in the position of function»Slight wrist flexion, MCPs flexion, IPs extension».

3. Surgical drainage: is indicated if pus formation is evident from the start or if there is no response after 1 day intensive antibiotic therapy.

- Acute paronychia & distal pulp space infection can be drained using local ring anaesthesia (without adrenaline) at the root of the finger.
- To obtain a clear bloodless field, a pneumatic tourniquet is applied around the upper arm, the hand is elevated for a few mins & then the tourniquet is inflated.
- Appropriate skin incisions are used & pus is drained by a sinus forceps.
- Pus should be sampled for C&S.
- Soft rubber drains are preferred e.g. a piece of surgical glove.

4. Postoperative: elevation, physiotherapy & wound dressings.

Subcuticular & SC Whitlow

- It is subepithelial collection of pus.
- Drainage is done by excision of the insensitive roof without anesthesia.
- It may be part of collar-stud abscess with SC component, which requires anesthesia for its evacuation.
- A collar-stud abscess forms when pus trickles down the dermis forming another subdermal component if the roof is callous.
- A SC abscess may also form without a subcuticular component.



- Due to the use of the hand and its exposure to contamination, the frequency of hand infection is high among manual workers and house wives.

- Proper treatment and early interference is needed to preserve the function of the hand.

Classification

1. Cutaneous & SC:

- a. Pulp space infection.
- b. Subcuticular & SC whitlow.
- c. Web space infection.
- d. Paronychia.

2. Fascial spaces infection:

- a. Midpalmar space infection.
- b. Thenar space infection.
- c. Space of Parona infection.
- d. Hypothenar space infection.

3. Synovial sheaths:

- a. Acute digital tenosynovitis.
- b. Ulnar bursitis.
- c. Radial bursitis.

4. Bone & joint infections.

Infections Related To The Nail

- Peri-ungual infection = Paronychia

I. Acute paronychia:

- **Def.:** acute suppurative infection of the nail fold.
- **Incidence:** The commonest form of hand infection.
- **C/P:** Localized pain & tenderness to one side of the nail fold accompanied with small red swelling.
- **Complications:**
 1. Extension subungual. «Below the nail»
 2. Extension to pulp space.
 3. Lymphangitis and lymphadenitis.
- **TTT:**
 1. Pus can be drained using a fine scalpel to raise the nail fold & to incise the skin cap through which pus points.
 2. If there is a subungual extension, the related part of nail is excised to provide proper drainage.

II. Chronic Paronychia:

- Develops in the chronically wet nails of dishwashers.
- Associated fungal infection & nail deformities are common.
- **TTT:** Keep the hand dry, Antifungal ttt, Removal of the affected nail.

Pulp Space Infection

Anatomy of the pulp:

- This space lies anterior to the distal phalanx & contains fatty tissue.
 - It's divided into locules by multiple septa running from the skin to the front of the bone.
 - It's closed proximally by the insertion of the profundus tendon to the palmar surface of the base of the distal phalanx.
 - The digital artery divides in the middle segment of the finger into an epiphyseal branch which enter the articular end outside the pulp & a diaphyseal branch which traverses the pulp.
- Pathology of pulp space infection:
- CO is mainly Staph. & to a less extent Strept.
 - Route of infection: direct inoculation by a pinprick, extension from paronychia, extension from middle pulp space, extension from cuticular or subcuticular infection.
 - The proximal part of the bone is protected as it's supplied by the epiphyseal branch of the digital artery before it enters the space → complete regeneration can be obtained from the undamaged proximal part



Clinical Picture

1. Localized pain and tenderness over the distal pulp space.
2. Swelling which is indurated and red.

3. General manifestations of infection.
4. No tenderness over the finger proximally or in the palm (D.D. of tenosynovitis).
5. Range of painless movement in all the joints (D.D. of arthritis).



Complications

1. Osteomyelitis of the distal phalanx due to digital vessel septic thrombophlebitis.
2. Extension to the middle pulp space.
3. Teno-synovitis.
4. Arthritis.
5. Blood stream and lymphatic spread.



Treatment

Early surgical drainage

1. Do not wait for fluctuation.
2. The incision is sited directly over the tenderest point and in direction of Langer's lines.

Web Space Infection

Anatomy of the web:

- The heads of the metacarpals are attached together by the transverse palmar ligaments.
- The web space lies in front of this ligament & deep to the skin.
- The space communicates proximally with mid palmar space.
- Distally it communicates with proximal pulp space of the adjacent fingers.
- It is filled with fat & is crossed by the lumbrical ms.



Clinical Picture

1. The patient complains of hot, red, tender swelling at the site of a web.
2. The fingers cannot be approximated because adduction increases pain.
3. The movement of all joints of the fingers is free (D.D. septic arthritis).
4. No swelling in fingers (D.D. Teno-synovitis).



Complications

Extend to:

1. Mid palmar space.
2. Proximal pulp Space.
3. Tendon sheath.



Treatment

1. A transverse skin incision is made over the point of maximal tenderness or fluctuation, and then an artery or a sinus forceps open the SC tissue in a longitudinal direction (to avoid damage to the digital nerves & vessels), to enter the abscess cavity.
2. Vertical dorsal incision.

Fascial Spaces

Hypothenar Space Infection

Source of infection:

1. Direct inoculation.
2. Complication of teno-synovitis of the flexor tendon of the little finger.
3. Extension from the web between little and ring fingers.
4. Extension from proximal pulp space.

CO: Staph., Strept. ... etc.



Clinical Picture

1. Localized Pain, Tenderness, Hotness & Redness in the hypothenar region.
2. Swelling on the medial aspect with accentuation of the hand concavity.
3. Free range of painless movements in the IP & MCPs of little finger (D.D Septic arthritis, Teno-synovitis, Ulnar bursitis).



Complications

- Extension to web space, proximal pulp space, mid palmar space or tendon sheath.
- Lymphatic and blood stream infection.



Treatment

A Vertical incision at the site of maximal tenderness or along the medial border of the 5th metacarpal bone is done. The space is entered by a sinus forceps i.e. Hilton's method.

Thenar Space Infection

Source of infection:

1. Direct inoculation.
2. Extension from proximal pulp space.
3. Extension from web space.
4. Complication of tenosynovitis.



Clinical Picture

- Swelling in the thenar eminence which is hot, red & severely tender.
- Edema on the dorsum of the hand.



Treatment

- General principles are followed.
- Incision:
 1. Transverse incision at the web then the space is opened by a sinus forceps (Hilton's method).
 2. Vertical incision on the lat. aspect of back of the 2nd metacarpal. «Commonly used incision»
 3. An incision that is done along the medial side of the thenar eminence should stop 2cm distal to the distal wrist crease to avoid injury of motor branch of median n. that supplies the thenar ms.

Anatomy of Fascial Spaces of the Hand

- The palmar aponeurosis is triangular in shape. Its apex is proximal and into it is inserted palmaris longus. Distally it gives slips to be attached to the transverse palmar ligaments between the medial 4 fingers.

- The medial edge of palmar aponeurosis gives a septum, which dips backwards and is attached to 5th metacarpal bone. Medial to this septum is the hypothenar space containing short ms of little finger.

- Another septum extends from the lateral edge to the ant. aspect of the 3rd metacarpal. This septum separates laterally thenar space from mid palmar space. This septum also covers the adductor pollicis.

- Mid palmar space is further divided into a superficial compartment superficial to the tendons and deep to the palmar aponeurosis called subaponeurotic space. The space behind the flexor tendons is called retro tendinous space.

Mid Palmar Space Infection

- The space is bounded anteriorly by palmar aponeurosis, medially by medial septum separating it from the hypothenar space, laterally by the lateral septum separating it from the thenar space.
- Mid palmar space is further divided into subaponeurotic & retro-tendinous spaces by flexor tendons.

Source of Infection:

1. Web space infection.
2. Thenar and hypothenar infection.
3. Tenosynovitis.
4. Direct inoculation. «Puncture»



Clinical Picture

1. Marked swelling which obliterates the palmar concavity (Frog's hand).
2. The middle of the palm is hot, red and tender.
3. Dorsal swelling is much more apparent than in any other hand infection.
4. Pain on moving the fingers with free range of movement of these fingers.
5. Sometimes a subaponeurotic infection forms a subcutaneous locus by forming a hole in the palmar aponeurosis forming a collar-stud Abscess. It is important to exclude deep sub-aponeurotic infection when one is draining a subcutaneous abscess in the palm.



Complications

1. Infection of tendon sheaths.
2. Spread to the web.
3. General spread.



Treatment

when pus is formed

- If there is a collar-stud abscess, incise along one of the hand creases, evacuating the SC abscess. The hole in the aponeurosis is explored and is enlarged to drain sub-aponeurotic collection.
- A transverse incision is done like that of a web space and by Hilton's method a sinus forceps is introduced into the retrotendinous space to drain pus.

Acute Tenosynovitis

«Most serious hand infection»

Surgical anatomy of the tendon sheathes:

1. The middle 3 fingers are surrounded by 3 tendon sheathes which extend from the distal phalanx to the level of the head of the corresponding metacarpal bone.
2. Tendon sheath of the thumb extends in the palm & lower part of forearm forming the radial bursa.
3. Tendon sheath of the little finger communicates with the sheath surrounding the flexor tendons in the palm. The point of meeting between the sheath of the little finger & the common flexor sheath is called (Kanavel point). The common flexor sheath extends up to the lower part of the forearm.

Infection Of Parona Space

- This space lies in the distal part of the forearm between the pronator quadratus & the flexor muscles. Drainage should be along the ulnar side of the forearm.

Source of infection:

1. Direct inoculation by pricks & wounds. «Usually»
2. Extension from a nearby infection:
 - Pulp space infection.
 - Web space infection.
 - Mid palmar space infection.
 - Arthritis and Osteomyelitis.
3. Blood stream infection.



Complications

1. Sloughing of the tendon due to thrombophlebitis of tendon vessels that run along the vinculae → stiff useless finger.
2. Spread to Mid palmar space & Space of parona.



Clinical Picture

1. History of the causative agent.
2. General inflammation manifestations: FAHM, Tachycardia, Constipation & White-coated tongue.
3. Swelling:
 - There is symmetrical enlargement of the palmar aspect of the finger extending from distal phalanx to the head of the metacarpal bones. The finger will be in the semi-flexed position.
 - The swelling in case of little finger extends in the middle of the palm & lower part of forearm. «Ulnar bursitis»
 - Swelling of the thumb extends in the thenar eminence & lower part of forearm. «Radial Bursitis»
4. Tenderness over the tendon sheath but maximal at the cut-de-sac. «At the distal phalanx & at the head of the metacarpal bone»
5. In the little finger the tenderest point is the Kanavel point. This point is between the 2 transverse palmar creases opposite the head of the 5th metacarpal bone.
6. Pain ↑↑ on moving the finger & is exaggerated by passive finger extension.
7. Semiflexion & Limitation of movement of the medial 4 fingers.
8. Radial bursitis gives a similar picture in the thumb.



Treatment

1. Early proper diagnosis.
2. Full antibiotic therapy.
3. Lack of response to intensive conservative ttt is an indication for surgery.
4. The affected sheath drained through 2 incisions at its proximal & distal extremities. A fine catheter is inserted through each incision to allow continuous drainage & irrigation of sheath with an antibiotic solution.

Diseases of Muscles, Tendons & Fascia

Carpal Tunnel \$

- o It's the commonest type of nerve compression \$.
- o The carpal tunnel is formed by the flexor retinaculum anteriorly & the distal row of the carpus posteriorly. • The median nerve & flexor tendons pass through it.
- o Swelling of synovial sheaths of flexor tendons ↑↑ the bulk of the contents of the tunnel compressing the median nerve.

Aetiology

- This disorder affects essentially the middle aged females.
- Precipitated by: Rheumatoid arthritis involving the synovial sheaths → ↑ tension under retinaculum, myxedema, pregnancy or acromegaly.
- May be idiopathic.

Clinical Picture

• Early manifestations

1. Pain in the thumb & lateral three fingers that gets worse by night.
2. Pain is increased by fully flexing the wrist.
3. The little finger is never affected.
4. The palm is not affected as the palmar branch of median nerve escapes compression.
5. Relief of pain is by injecting the tunnel with lidocaine & CS & by splinting the wrist at night.

• Late manifestations

1. Paraesthesia & altered sensibility of skin supplied by the median nerve.
2. Weakness & atrophy of the thenar eminence muscle.

Treatment

- Longitudinal incision of flexor retinaculum.
- After decompression, the median nerve should be inspected & if it still shows a constriction → release its epineurium.

Dupuytren's Contracture

Definition

- Idiopathic disorder known as palmar fasciitis.
- It's commonly confused with hand pain produced by cervical spondylosis.

Incidence

- Males are affected 10 times more than females.
- There is higher incidence in cirrhotics, alcoholics, diabetics & epileptics under phenytoin ttt.

Aetiology

Idiopathic but some cases are familial.



Pathophysiology

Progressive thickening & contraction of the palmar aponeurosis.



Clinical Picture

- Bilateral in 1/2 of cases.
 - It affects mainly the medial side of the palmar aponeurosis.
1. It starts as a nodule at the base of the ring or little finger followed by contracture which throws the finger into flexion.
 2. The skin gets adherent to the fascia & contracts.
 3. The other fingers progressively become involved.
 4. Later, the capsules of MCPs & PIPs joints become involved & affected by the flexion deformity.
 5. The DIPs are free.
 6. Palpation of the palm → firm irregular nodule, 1-2 cm proximal to the base of ring finger.



Treatment

- **Early cases** → physiotherapy.
- **Late cases** → surgery which varies from minor SC fasciotomy to extensive operation including:
 1. Excision of the aponeurosis.
 2. Joint capsulotomy.
 3. Skin grafting to cover the resulting defect.

Simple Ganglion

- It is a cystic swelling related to a joint or tendon sheath, occurring mostly on the dorsum of wrist & foot.
- The cyst contains clear gelatinous fluid and its wall consists of fibrous tissue.
- It is either due to herniation of synovial membrane through tendon sheath or mucoid degeneration predisposed to by trauma.



Clinical Picture

- Rounded cystic swelling, which is painless & not attached to skin.
- It is translucent, movable when tendon is relaxed & becomes fixed on contraction.



Treatment

1. SC rupture by pressure.
2. Puncture followed by pressure.
3. Puncture followed by injection of hyaluronidase.
4. Excision through a transverse incision.

Compound Palmar Ganglion

- o This is TB synovitis of the synovial sheath of the fingers under the flexor retinaculum. It contains TB granulation tissue, free fluid and melon seed bodies (fibrinous).



Clinical Picture

- A fluctuating swelling in the lower part of forearm & palm with a band like constriction at the level of the flexor retinaculum.



Treatment

1. Anti-TB ttt.
2. Immobilization in plaster of paris.
3. Surgical excision: Complete excision is not recommended & incision with evacuation of contents is preferred.

Volkmann's Ischemic Contracture



Definition

Fibrosis & shortening of the ms of the front of the forearm due to acute ischemia & infarction.



Aetiology

- Brachial artery compromise after injuries around the elbow joint → acute ischemia.
 1. Supracondylar fracture in children: the ischemia is more commonly due to a tight plaster cast rather than due to the fracture itself.
 2. Other less frequent causes as direct brachial artery injury or arterial embolism.
- The acute ms ischemia → infarction after 6-12 hrs.
- Ms fibrosis & shortening develop within a few months.



Clinical Picture

• Early:

1. Manifestations of acute ischemia: Pain, Pallor, Paralysis, Parasthesia, Pulselessness.
2. Appearance of these features in a pt with fracture or after application of a plaster cast is very serious as this stage of ischemia can be corrected & the ms saved.

• Later:

1. The pt will present with the typical deformity of: flexion at the wrist joint, extension at the MCPs, flexion at all the IP joints. Extension of the wrist → more flexion at the IP joints. With flexion at the wrist, the surgeon can passively extend the finger (unlike the Dupuytren's contracture).
2. An important diagnostic feature is that all ms have some functions.



Treatment

1. Prophylactic ttt: If the pt develops acute ischemia with the 5(Ps), the plaster is removed, the fracture is recorrected, and the pt is given vasodilators. If no immediate response is obtained, the vessel is exposed and dealt with acc. to the type of injury present.

2. In established case:

- a. Early: physiotherapy by gradual stretching & splinting.
- b. Late: (Ms slide operation): the common flexor origin is detached from the med. epicondyle & is fixed at a lower level.



D.D. Of Claw Hand

1. Volkmann's ischemic contracture.
2. Combined median & ulnar nerve injuries.
3. Lesions affecting the lower roots or medial cord of the brachial plexus e.g. Klumpke's paralysis, malignant infiltration of the brachial plexus.
4. Advanced R.A.
5. Spinal cord lesions as syringomyelia & poliomyelitis.

Chronic Tendonitis

1. Tennis elbow:

- **Cause:** direct trauma or repeated athletic activity.
- The pt complains of pain in the elbow at rest & in particular when he uses the hand (extensors).
- Pain develops at the site of attachment of the extensor ms of forearm to the lateral epicondyle.
- **TTT:** rest. In resistant cases → local injection of CS & anaesthetics.

2. Rotator cuff (Supraspinatus) tendonitis:

- The commonest cause of shoulder pain with limitation of movement.
- **Cause:** repeated trauma from sports or occupational activities.
- Inflammation often extends to affect the subacromial bursa.
- Active abduction is particularly painful when the shoulder moves bet. 60-120 degree → painful arc \$
- **TTT:**
 - a. Conservative using NSAIDs.
 - b. Shoulder immobilization for few days followed by gradual active exercise to restore the full range of shoulder movement.
 - c. Resistant cases → local injection of CS-lidocaine mixture.

3. Stenosing teno-vaginitis:

- **Cause:** fibrous stricture in a tendon sheath.
- There are 2 common examples & both are in the hand.

Chronic Bursitis

- Bursae are fluid filled cavities lined by flattened epithelium.

Types:

A. Anatomical bursae:

- Are normally found to allow easy movement between tendons, bones, and skin.
- Chronic inflammation of such bursae usually results from repeated friction & presents with mildly painful cystic swellings.

- Common sites:

1. Prepatellar bursitis (House-maid's knee).
2. Infrapatellar bursitis (Clergyman's knee).
3. Olecranon bursitis (student's elbow).
4. Semimembranous bursitis:

- The bursa lies in the popliteal fossa between the medial head of gastrocnemius & semimembranosus tendon and frequently communicates with the cavity of knee.

- The bursa is rounded fluctuating swelling, becomes tense on extension & flaccid on flexion of the joint.

- **TTT:** careful dissection & excision.

B. Adventitious bursae:

- Develop in any site of friction between 2 layers of tissues. A known example is «bunion» which develops bet. the skin & head of the 1st metatarsal, particularly with the hallux valgus deformity.
- If friction can be abolished, bursitis may regress, otherwise the ttt is excision & correction of any underlying deformity.

Ingrowing Toe Nail



Introducation

- A common problem in which a sharp edge of the big toe nail ingrows in the adjacent skin fold.
- Young males predominate.



Aetiology

1. Faulty nail trimming. Oblique trimming of the nail sides may leave behind a sharp spike that starts the condition.
2. Tight shoes.

Complications: infection commonly sets in & the condition may worsen by attempts of the pt to cut away the nail.



Treatment

A. Conservative ttt indicated in early cases :

- The aim is to lift off the nail spike.
- A piece of gauze soaked in an antiseptic is inserted beneath the ingrowing part of the nail to raise it up & to ease away the injured tissues.
- The piece of gauze is changed daily until the nail grows.
- The pt is instructed to avoid tight shoes & shown the proper technique of square nail trimming.

B. Surgery: Indicated for failure of conservative ttt & for suppurative cases.

- Simple avulsion of the nail provides rapid relief of severe suppurative infection but recurrence is common with regrowth of the nail.
- Wedge (segmental) resection of the germinal matrix is the definitive ttt.
- Position: on the back.
- Tourniquet: on toe near its base to provide a clear bloodless field.
- Anesthesia: General or local (without adrenaline).
- A longitudinal incision is made through the affected side of the nail down to the bone & extending proximally to the nail root.
- Another incision is made through the skin by the side of the lesion down to the phalanx
- The wedge of tissue between the 2 incisions is excised, leaving bare bone without any remnant of the germinal matrix.
- The gap may be left open to granulate or may be closed with 2 or 3 interrupted sutures.

Swellings Of Popliteal Fossa

A. Cystic swellings:

1. Skin & S.C. tissue: Abscess, hemangioma, lymphangioma.
2. Vein: Saccular varicosity of upper end of short saphenous vein, V.V.
3. Artery: Popliteal aneurysm, arterio - venous fistula.
4. Semimembranosus bursitis.

B. Solid swellings:

1. Skin & SC tissue: Upoma, neurofibroma.
2. Muscles: Fibrosarcoma.
3. Nerves: Neurofibroma.
4. Periosteum: Fibrosarcoma.
5. Bones: Acute and chronic osteomyelitis, osteoclastoma, osteosarcoma, secondaries of lower end of femur or upper end of tibia.

Soft Tissue Sarcomas



Definition

Malignant tumors arising in the extra-skeletal C.t.



Aetiology

Not exactly known:

1. Following irradiation for other malignancies as Hodgkin's lymphoma.
2. Lymphangiosarcoma may develop in the chronic post mastectomy arm edema (Stewart- Treves disease).
3. Neurofibrosarcoma may develop in pts with Von Recklinghausen's disease.



Pathophysiology

(Types)

- Most of them arise from primitive multipotential mesenchymal Ct cells which differentiate into one of the following:

1. Malignant fibrous histiocytoma.
2. Liposarcoma.
3. Rhabdomyosarcoma.
4. Synovial sarcoma.
5. Malignant nerve sheath tumor.
6. Leiomyosarcoma.
7. Fibrosarcoma.
8. Angiosarcoma.



Gross

- There is a well-defined pseudocapsule. Enucleation of the tumor from within the pseudocapsule is inadequate.
- The cut section is fleshy & shows areas of necrosis & hemorrhage.



Spread

Direct, blood & lymphatic spread.



Clinical Picture

1. Swelling.
2. Pain & disability are often absent.
3. Spread.



Investigations

1. Open biopsy.
2. CT & MRI.
3. Investigations to detect spread.



Treatment

1. Operable cases:

- A combination of radical surgical excision & adjuvant radiotherapy is the standard ttt. The extent of surgery depends on the extent of tumor invasion.
- Major reconstruction of skin & soft tissue defects is commonly required.
- If radical resection is judged to leave a useless limb, amputation is indicated.

2. Inoperable cases: Palliative surgical excision if possible.

Skin & SC tissues

Sebaceous (Epidermoid) Cyst



Definition

A retention cyst due to blockage of the duct of a sebaceous gland.

Structure:

- It is lined by str. sq. epithelium & contains a foul smelling, white material composed of keratin, epithelial cells & granular debris.



Clinical Picture

1. Age: Slowly growing so, rarely seen before adolescence.

2. Site: Most commonly in the scalp, face, neck or scrotum but they can occur anywhere except the palm of the sole of the foot, which are devoid of sebaceous glands.

3. Ex.:

- Small (less commonly large), well defined, cystic swelling, which is usually attached to the skin at one point, which is the site of the duct.
- A punctum may be seen.



Complications

«Only infection is common»

1. Infection: Cyst becomes red, painful & tender and an abscess may form. It may subside by antibiotics, otherwise if abscess forms, it should be drained & the wall of the cyst curetted. Infection makes the cyst more difficult to excise as it becomes adherent to the surrounding SC tissues.

2. Sebaceous horn: The contents of the cyst may come out slowly and become inspissated in successive layers over the base.

3. Ulceration: An infected cyst may undergo ulceration leading to the appearance of an ulcer with raised edges. This ulcer is called (Cock's peculiar tumor) and it may be mistaken for a carcinoma, but it lacks the induration of malignant lesions. If in doubt a biopsy should be taken.

4. Localized alopecia.



Treatment

- Complete excision of the cyst with an ellipse of overlying skin containing the punctum.
- The operation can be done under local anesthesia if the cyst is small.

Dermoid cyst



Definition

- Cyst lined by str. Sq. epithelium & contains sebaceous material. Sometimes hairs or sebaceous glands may grow from the wall of the cyst.

Types	Due to	Sites	Structure
1. Sequestration dermoid	SC inclusion of portions of the surface epithelium along the lines of fusion of cutaneous segments during fetal life. They may not appear clinically except after few years when the cyst begins to distend.	e.g. external angular dermoids at the outer angle of the eye, at the root of the nose, around the ear or along the midline of the body.	A well-defined, globular, cystic swelling, which is not attached to the skin. The underlying bone may be hollowed out and there may be a pedicle connecting the deep aspect of the cyst to the dura matter.
2. Tubulodermoids	Distension of remnants of embryonic ducts such as branchial & thyroglossal cyst		
3. Inclusion dermoids	Inclusion of the epidermis during closure of a cavity.	Sublingual, Suprasternal, Intracranial, Intrapinal.	
4. Teratomatous dermoid	These are benign forms of teratomas.	Mostly in the ovary but are occasionally found in the testis or post. mediastinum.	The cyst is lined by sq. epith. but contains teeth, hairs, bone, cartilage or glands.
5. Implantation dermoids	Puncture wounds which displace some epithelial cells into the SC tissues. The displaced cells retain their viability & form a dermoid cyst.	Mainly in the fingers, palm or sole.	Usually small, tense. Overlying skin is sometimes scarred



Treatment

Surgical excision.



1. In children with a dermoid cyst in the scalp it is better to wait until closure of the skull sutures as some cysts may communicate with the dura.

2. Surgery for dermoid cyst is more difficult than that for sebaceous cyst as the dermoid is deeper.

[1] Hemangioma

(previously known as strawberry hemangioma)

1. It affects 10% of white infants & F:M = 3:1.
2. It presents by three stages: proliferation, involution & involuted phase).

[2] Capillary malformation

(port-wine stain)

	1. Hemangioma	2. Capillary malformation
	(Strawberry angioma)	(Port wine stain, Nevus flammeus)
Ex.	The lesion is red in color and slightly raised above the surface appearing after birth & increasing in size in the following months (stage of proliferation).	The lesion is dark purple in color & is not raised above the surface. Pressure causes blanching but the color returns immediately after release of pressure.
Site	The commonest site is the head & neck.	May take the distribution of one of the branches of the trigeminal nerve, but the lesion does not cross the midline. Sometimes, a port wine stain of the face is associated with similar lesions in meninges (Sturge-Weber \$).
Natural history	After the age of one year, the lesion starts to undergo involution. Eventually the color fades & flattening occurs so that there may be complete involution at the age of 10 years. This course is not predictable & sometimes, the lesion continues to grow.	This lesion is present since birth and it does not undergo involution.
problems	• The peri-orbital one may encroach on the visual field → amblyopia. • Parotid hemangioma may → auditory canal obstruction. • Large cutan. or visceral may → CHF. • Large visceral hemaniomata may → entrapment of platelets → thrombocytopenia.	
TTT	(a) Reassurance & observation as the skin appearance after involution may be better than the scar following excision. (b) If problems occur: • Oral or intra-lesional CS • Laser or photocoagulation • Surgical debulking.	(a) Laser ablation. Pulsated dye laser in children, YAG in adults with less favorable results than in children. (b) Surgery & grafting may be difficult as the lesion may involve a large area of the face.

Vascular Anomalies

1. Hemangioma.

2. vascular malformations:

- Low flow lesions

- a. Capillary malformation
- b. Venous malformation

- High flow lesions

- a. Arterial
- b. Arteriovenous.

- Lymphatic malformations.

N.B: differentiation between high and low flow lesions is done by duplex & MRA.

[3] Venous malformations

previously known as cavernous hemangioma



Clinical Picture

Presents as a compressible swelling with some discoloration of the overlying skin.

Site:

- Occur in the SC tissue or the submucous lining of the cheek, lips or tongue. It may involve the internal organs as the liver.

Natural history: The lesion usually appears later than hemangioma & it has no tendency to involution.



Complications

1. It may lead to thrombocytopenic purpura due to sequestration of a large number of platelets.
2. Atrophy of the overlying skin may lead to severe hge.
3. Septicemia from invasion by microorganisms.



Treatment

1. Laser therapy.
2. Injection of sclerosing solutions as hypertonic saline, 100% alcohol, Na tetradecylsulfate.
3. Surgical excision.



Compression therapy for venous malformations may relieve pain & edema.

[4] Arterial Malformations

(Cirroid Aneurysm)



Clinical Picture

The lesion appears as soft, compressible & pulsating swelling with a marked bruit.

Site:

- It occurs most commonly in the scalp especially in the temporal or occipital regions & may involve the underlying bone.

Nature: Abn. development of arterial structures including stenosis, hyperplasia, duplication &/or tortuosity.

Complications: Ulceration of overlying skin may lead to serious hge, as the bleeding is arterial.



Treatment

- Difficult as the swelling is supplied by multiple feeding arteries which have to be ligated. Preliminary embolization of the feeding vessels may be tried.

[5] A-V malformations

- It is high flow lesions with abn. connections bet. arteries & veins without intervening capillary bed
- It may present at birth but may not be evident until late childhood.
- It may be localized or diffuse. It manifests as localized swelling with ↑↑ surface temperature.
- It may results in growth disturbance or skeletal distortion. There may be local gigantism.
- There may be palpable thrill, murmur.

• TTT:

- a. Feeding vessel ligation/Embolization often → rapid enlargement of collateral vessels → ↑↑ Lesion size.
- b. Preoperative angiography & feeding vessel embolization followed by excision next day is done. Extensive coverage with a free flap may be needed.

Hereditary hemorrhagic telangiectasia: AD disease characterized by multiple cutaneous, visceral & mucosal AVMs.

[6] Lymphatic malformations

(see cystic hygroma).

- The commonest cause of congenital macroglossia, macrocheilia (lip enlargement) & macrotia.

TTT:

1. Percutaneous aspiration followed by sclerosis may provide short term improvement.
2. Surgical excision often requires multiple staged procedures. It's technically easier as child grows: consider waiting until at least 3 years of age.

Lipoma



Definition

Benign tumor of the fatty tissue.



Incidence

Common in adult, single or multiple.



Classification

A. According to composition:

1. **Ordinary lipoma:** Mainly fat with little fibrous tissue. Soft.
2. **Fibrolipoma:** Contains excess fibrous tissue rendering it firm.
3. **Angiolipoma:** Contains angiomatous tissue rendering it bluish.
4. **Myxolipoma:** Contains myxomatous tissue.

B. According to presentation: Lipomas may present as:

1. **A solitary swelling.**
2. **Multiple lipomatosis:** in which the limbs or the trunk are the seat of multiple lipomas (D.D of multiple swellings).

3. Diffuse lipomatous deposits: These can occur in certain areas, e.g. pts with myxedema have supraclavicular fatty deposits, elderly persons may develop lipomatous deposits below the chin & sometimes females may develop painful fatty deposits in the thigh (Dercum's disease).

C. According to the site of origin: Solitary lipomas are classified into:

1. SC lipomata: These are the commonest and they have the following characters:

- Slowly growing tumor in the SC tissue.
- The tumor is painless and is not tender.
- Lobulated surface, which may be attached to the skin at multiple points. Yellowish in color.
- The consistency is soft, although some lipomata may give pseudofluctuation; this is due to mobility of the tumor in its bed & as fat at warm temperature may undergo liquefaction.
- It has a well-defined slippery edge due to movement of the tumor inside its capsule.
- The swelling is mobile over deep structures.

2. Subfascial lipomata: Occur under the deep fascia. Difficult to diagnose. Not attached to the skin. No slippery edge.

3. Intermuscular lipomata: Difficult to diagnose and has to be differentiated from a fibrosarcoma. The latter is hard in consistency and grows rapidly. Disappears when the ms above contracts.

4. Submucous lipomata: A submucous lipoma in the larynx may cause respiratory obstruction. A submucous lipoma in the intestine may initiate intussusception causing intestinal obstruction.

5. Parosteal lipomata: Arise in relation to cranial bones & cause erosion of the underlying bones.

6. Extradural lipomata: Are sometimes found within the spinal canal & may cause paraplegia (never in the skull, no fat).

7. Intra-articular lipoma.

8. Retroperitoneal lipoma.



Complications

Are rare and include:

- Degenerative changes → liquefaction & calcification.
- Malignant transformation is very rare, but it can occur only in a retroperitoneal lipoma.
- Submucous lipoma & extradural lipoma (see before).



Treatment

Surgical excision of SC lipoma for cosmetic reasons by enucleation from inside its capsule (enucleation).

Molluscum Sebaceum (Keratoacanthoma)

- Supposed to be due to sun exposure.
- Occurs commonly in the face of middle aged persons.
- Made of keratinizing squamous cells which resolve spontaneously.
- Mimics epithelioma, Rodent ulcer → Unnecessary surgery.

- Firm, Rounded, Reddish papule, enlarging rapidly over 8 w → Rounded, Slightly umbilicated mass 1-2 cm in diameter. The center contains a horny plug covered by crust concealing a keratin filled crater
- Heals by shedding of the horny core spontaneously in about 6 months

Callosity

- Area of superficial skin thickening due to continuous friction and pressure, usually not painful.
- On the toes & sole from wearing ill-fitting shoes.
- Could also be seen on the fingers & hands of manual workers.
- Circumscribed, yellowish white plaque.
- Could be infected → Abscess.
- **TTT:** the pt should avoid the causative agent & the callosity should be shaved with a razor blade & repeatedly painted with an ointment containing salicylic acid.

Corn

- Similar to callosity but caused by pressure as well, compressing the sensory nerve endings → much pain with similar ttt. Excision & suture are rarely necessary .

Wart

Viral infection in an abrasion → localized overgrowth of the epidermis & papillae of the skin.

It is small horny projection & may be multiple. Its ttt includes:

1. Curettage & diathermy.
2. Repeated application of glacial acetic acid.
3. Cryosurgery by liquid Nitrogen or NO.

Papilloma

- Benign tumor, usually pigmented commonly develops in large numbers in the face, arms & upper trunk
- May be familial (AD),
- Malignant transformation is rare.
- TTT is excision, cryosurgery or cautery.

Melanocytic Tumors

o Melanocytes arise from the neural crest & lodge in between the basal cells of the epidermis. Various hamartomas or true tumors can arise from the melanocytes.

Benign lesions of melanocytes:

1. Lentigo: Melanocytes replace the basal layer of the epidermis over a certain area. Clinically: It appears as a spot varying in color from pale brown to black.

2. Junctional-pigmented nevus: More proliferation of melanocytes at the junction of epidermis & dermis. Clinically: Looks like a lentigo.

3. Compound pigmented nevus: Some melanocytes pass to the dermis after disrupting the BM: These melanocytes lose their capacity to form melanin & they are called nevus cells. Clinically:

- o Lesions are raised above the surface and vary in color from pale brown to black.

- o Sometimes dark hairs are seen growing from the surface of the lesion. Rarely, an extensive area of the skin is replaced by such hairy-pigmented nevus (Giant hair nevus).

4. Intradermal pigmented nevus: (Commonest). Junctional activity ceases around puberty in most cases & nevus cells in the dermis undergo maturation.



Clinical Picture

They look like compound pigmented nevus.

Can pigmented nevi turn malignant?

1. The giant hairy-pigmented nevi can give rise to metastases after turning malignant.

2. After puberty any pigmented nevus with junctional activity has the potential to undergo malignant changes, but this is rare.

Indications for surgical excision:

1. For cosmetic reasons.

2. If they are subjected to repeated trauma e.g. during shaving.

3. If there is suspicion of malignant transformation.

Adequate surgical excision should be performed & the specimen should be examined histologically

Precancerous skin lesions

Squamous keratosis (actinic or senile):

- Present by dry, rough, inelastic & irregular pigmented areas of skin with hyperkeratosis. It is the most important precursor of sq. cell carcinoma as there's pleomorphism, ↑↑ mitosis & irregular cell differentiation under the microscope.

Bowen's disease:

- Arises in non exposed skin with sharply demarcated, rounded reddish patches which enlarge slowly with marked cellular hyperplasia. It's usually treated as psoriasis.

Melanoma



Incidence

- In Western countries, the incidence of malignant melanoma is increasing, which may be due to defective ozone layer. Malignant melanoma is almost unknown before puberty.

+ Aetiology

1. Prolonged exposure to ultraviolet rays of the sun.
2. Albinism & xeroderma pigmentosa.
3. On top of a benign nevus (discuss). Criteria of malignant transformation of a benign nevus:
 - a. ↑ Size or thickness.
 - b. ↑ Pigmentation or depigmentation.
 - c. Ulceration & bleeding, itching, tingling.
 - d. Development of satellite nodules.

Items	Superficial spreading melanoma	Nodular melanoma	Lentigo-maligna melanoma (Hutchinson's melanotic freckle)	Acral lentiginous melanoma	Amelanotic melanoma
Incidence	64%	12-25 %	1-15 %	Rare	Rare
Site	In any part of the body.	In any part of the body.	Usually in the face.	In the palms & soles.	It may occur beneath the nail.
Age	Usually in middle age	In younger age	Elderly		
Clinically	1. Raised above the surface. 2. Edge: Irregular. 3. Spread along the epidermis.	1. Raised above the surface. 2. Surface: Smooth. 3. Color: Gray or Black. 4. Liable to ulceration.	1. Some areas may regress. 2. Flat, Brown, Slowly growing macule. 3. Spreads along the basal layer of epidermis.		It is not pigmented.
Prognosis		Bad as malignant cells invade dermis.	The least malignant.	Poor.	Very poor.

- Malignant melanoma may rarely arise in the eye, in the meninges, or at the mucocutaneous junction as the anal canal.

Prognostic Factors, which have a major influence on the prognosis, are:

- A. The thickness of the tumor (Breslow classification).
- B. The depth of invasion of the skin (Clark's level of invasion) as follows:
 1. Epidermis.
 2. Dermo-epidermal junction.
 3. Superficial papillary dermis.
 4. Deep papillary dermis.
 5. SC tissue.

✳ Spread

- 1. Direct:** To SC tissues & the deep fascia.
- 2. Lymphatic:** by permeation or embolism. Lymphatic permeation leads to satellite nodules around the tumor or between the tumor and the regional lymph nodes.
- 3. Blood:** Malignant melanoma can spread to any organ. 2ry deposits are usually black.



Differential Diagnosis

1. Granuloma.
2. Pigmented basal cell carcinoma.
3. Hemangioma.
4. Compound or junctional nevus.



Investigations

- The only sure method of diagnosis of malignant melanoma is Biopsy & Histological Ex..
- The biopsy should include the whole skin & SC tissue with safety margin 3mm at least.
- Paraffin section is better than frozen section.



Treatment

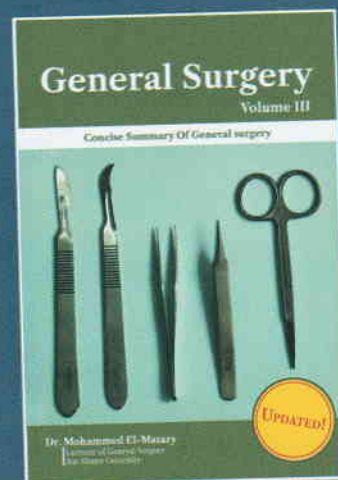
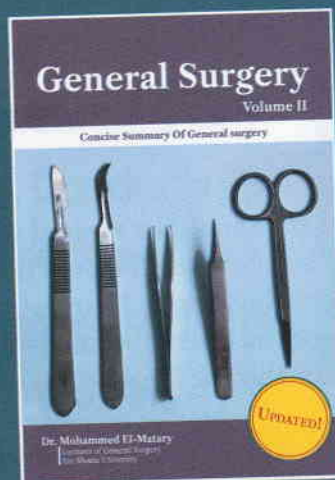
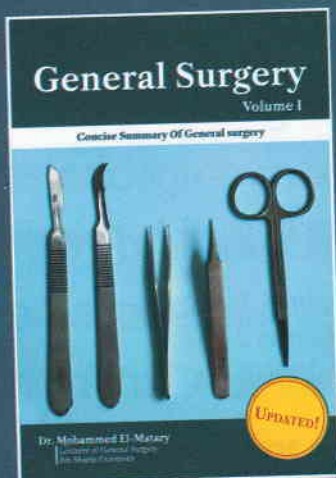
1. The 1ry lesion: Should be excised with a safety margin 2-3 cm together with the SC fat but not including the deep fascia.

- o If the tumor thickness <1mm → safety margin is 1 cm.
- o If the tumor thickness 1-4mm → safety margin is 2 cm.
- o If the tumor thickness > 4mm → safety margin is 3 cm.

2. The LNs:

- o If the regional LNs are enlarged & firm, a radical block dissection is performed.
- o If the LNs are not frankly malignant on clinical examination, FNAC is performed.
- o Prophylactic block dissection is no longer performed.

3. Metastases: Are treated by chemotherapy, INF & IL-2.



Mohammed El-Matary M.D

Assistant Professor of General Surgery at Ain Shams Univeristy. Working in the academic teaching and practice field since 1986. Supervising many researches of Master degree candidates. Conducting written, oral and clinical assessment exams for evaluation of Master and MD candidates. Author of different textbooks in different surgical fields covering all surgical aspects.



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